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THE CEREBROSPINAL FLUID IN 'ESSENTIAL' EPILEPSY*

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The data in the literature dealing with the cerebrospinal fluid of patients with epilepsy are unsatisfactory. Most of the reports present the incomplete findings of a few cases. Also, many authors have made no attempt to separate the cases in which the convulsions are associated with inflammatory, expanding or other lesions of the brain from the cases in which there is no apparent cause for the seizures. Neel [1] examined the fluids of 381 cases with convulsive seizures. In 129 of these cases the cause of the convulsive seizures was unknown. In this group he found that 11 per cent. had more than six cells and 14 per cent. had an increased protein content. This is in marked contrast to the percentage of abnormalities in the group of cases with convulsive seizures of known cause. In this group 69 per cent. had an increase in the cell count of the fluids and 75 per cent. an increased protein content. Patterson and Levi,[2] in their 50 cases, measured pressure with patients in the unorthodox sitting position and their measurements of pressure cannot be used. Twenty-two per cent. of the fluids had five or more cells per c.mm.; 10 per cent. had a positive globulin test; the sugar and chloride concentration was normal in the 10 examined cases. Marchand and Courtois,[3] in 100 cases of 'essential epilepsy,' measured the pressure (horizontal position) in 17 patients, in three of whom it was above 250 mm. of water. In 19 per cent. the protein was more than 30 mg. The highest cell count was 6 per c.mm. Kulkow,[4] in 50 cases, found an increased pressure in half of the cases. The cell count was increased in four cases and the globulin in 22. The sugar content was determined in 15 cases and found to be normal. Fay [5] reported the range of cerebrospinal fluid pressure readings

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in 100 cases of epilepsy. Pressures were measured in both the horizontal and vertical positions. In the horizontal position his results varied between 3 and 20 mm. of mercury (40 to 270 mm. of water) with an average of 10·5 mm. of mercury (142 mm. of water).

PERSONAL RESULTS

We here report the results of lumbar puncture of 810 patients having recurrent seizures. The great majority of these were seen by us during the past 12 years as private or hospital patients. These patients had as a routine a carefully taken history, physical and neurological examinations, roentgen-ray films of the skull and examinations of the blood and the cerebrospinal fluid. Therefore, practically all patients with a presenting symptom of seizure had lumbar puncture, but this report deals only with that portion of the total group who, on discharge, did not receive a diagnosis of organic nervous disease. Patients with seizures associated with abscess or tumour of the brain, syphilis of the nervous system, meningitis, encephalitis, multiple sclerosis, cerebral trauma, birth injury, cerebral arteriosclerosis, cerebral vascular lesions, chronic nephritis, uræmia or acute infections are excluded. We realize that it is impossible on the basis of an examination short of the postmortem table to exclude with accuracy all such cases. This is especially true of cases with brain tumour, since convulsive seizures may precede the onset of the other symptoms of a brain tumour by several or even many years.

When we speak of the 'essential' epileptic, we use that term not as a diagnosis, but simply as a convenient means of indicating patients whose condition is as yet undiagnosed. This elimination of patients with organic nervous disease was based chiefly on the history and findings on examination and in part on the results of the spinal fluid examinations. In many instances an abnormal spinal fluid finding was the principal or only evidence that led to the establishment of the diagnosis of central nervous system pathology. This was particularly true with cases of syphilis of the nervous system or tumour of the brain. Hence, this report views a group of patients after a winnowing process and not before. This fact should not be forgotten by the physician who is considering whether his patient should have a lumbar puncture performed. The previous reports from this laboratory[6, 7, 8, 9] dealing with a portion of the cases here reported did not attempt segregation of diagnosed and undiagnosed groups.

Pressure of the Cerebrospinal Fluid.—In considering the question of ætiology of seizures, abnormality in intracranial pressure is of considerable importance. Animal experiments have suggested that convulsant drugs more readily produce convulsions when intracranial pressure is high. Increase of pressure has been blamed for the convulsions associated with cerebral neoplasms, but against this is the fact that in these cases convulsions may occur early in the course of the disease before the intracranial

pressure is increased and also that seizures are relatively infrequent in patients with posterior fossa tumours, in which pressures are usually very high. Seizures which follow forcing of fluids and injection of pitressin are supposedly related to the retention of fluid and increased intracranial pressure. Fay [5] has proposed a 'hydraulic' theory of epilepsy based on the idea of a defective absorption of cerebrospinal fluid and consequent increase in the volume and pressure of the fluid. His measurements of pressure (made with a mercury manometer) are remarkably similar to ours, but his standard for normal pressures is lower than that used by us. Our standard of normal is based on our own experience with the method used in our neurological service over a period of years.

Measurements of pressure were made by means of the Ayer manometer or the Fremont-Smith modification. Patients were punctured in the lumbar region while lying in the lateral recumbent position. Readings were delayed until the patient was relaxed and the spinal fluid in the manometer had reached a steady state. Pressures obtained by this method were considered normal if between 70 and 180 mm. of cerebrospinal fluid; questionable if between 180 and 200 mm., and definitely abnormal if above 200 mm.

We tabulated the pressure at the initial puncture of 735 patients. The highest pressure was 300 mm.; the lowest 35 mm., and the average 143 mm.

The percentage distribution of pressure readings in these 735 patients is shown in Table I.

TABLE I. THE RANGE OF THE CEREBROSPINAL FLUID PRESSURE IN 735 CASES OF 'ESSENTIAL' EPILEPSY

Cerebrospinal fluid pressure (mm. of C.S.F.)	Number of cases	Percentage
35 to 70	17	2
70 to 100	71	10
100 to 150	343	46
150 to 180	182	25
180 to 200	57	8
200 to 250	59	8
250 to 300	6	1
	735	100

Eighty-one per cent. of the patients had a normal pressure. Some of the pressures in the neighbourhood of 200 mm. might be explained by the fact that in the early years of this investigation the importance of not having the patient's head and legs in a flexed position was not realized. Only six patients—less than 1 per cent. of the total—had a pressure above 250 mm.

These measurements were made in a period between seizures. Pressure

readings just before a seizure are important with reference to theories regarding the immediate cause of a seizure. A preliminary fall of pressure would lend support to the theory of vascular spasm, whereas an elevation would be in favour of an acute cerebral œdema or hydrocephalus or a vascular dilatation.

The senior author has on numerous occasions observed the spinal fluid pressure during a petit mal seizure. Before the attack, there was never any significant change in pressure unless the seizure was precipitated by hyperpnœa, in which cases the resulting alkalosis caused a constriction of cerebral vessels and a fall of pressure. During the petit mal attack, also, there was no change unless respirations were suspended long enough to produce venous congestion and a rise. On several occasions, he has observed the changes in pressure preceding and during grand mal convulsions. Whether there was a preliminary change in pressure depended on the point in the respiratory cycle at which the patient's tonic spasm began. When the glottis was closed, if the patient attempted to inspire, the pressure fell; if to expire, it rose. Observations made on successive days in one patient are shown in fig. 1. The fact that during a convulsion the intracranial pressure becomes as great as 700 mm. may help to explain the dilated ventricles and the cortical atrophy which so many chronic cases exhibit.

These observations indicate that changes in intracranial pressure at the time of seizure are passive rather than active factors in seizures. They are in agreement with the observations recently reported by Denny-Brown and Robertson,[10] who made graphic records of the cerebrospinal fluid pressures of six patients during convulsion.

White Corpuscles.—The white cells were counted in the fluids from 734 patients. The ordinary blood counting chamber was used, the cells being faintly stained with Unna's polychrome methylene blue. The number of cells varied between 0 and 21 per c.mm., with an average of 2 per c.mm. Only one patient had more than 10 cells per c.mm. and only 30 (4 per cent.) had more than five cells. As previously explained, cases believed to have any infection of the brain or meninges were excluded.

Protein Content.—The protein content of the fluid was measured in 793 cases. The Denis-Ayer [11] method was used and 45 mg. per 100 c.cm. was considered the upper limit of normal. The results varied between 7 and 200 mg., with an average of 31 mg. per 100 c.cm. The percentage distribution of these 793 values is shown in Table II.

Since there does not seem to be pathological significance in low values, 90 per cent. may be considered normal. In 10 per cent. the protein content was abnormally high, though in most instances the increase was moderate. The patient with the highest concentration of protein (200 mg.) one year later had 87 mg. and 10 years later 56 mg. per 100 c.cm. At autopsy 13 years after the first puncture there were several 'plaques jaunes' in the tips of the temporal lobes, probably the result of trauma received during some of his more than 10,000 convulsions.

FIG. 1.—Changes in cerebrospinal fluid pressure accompanying grand mal (G.M.) convulsive seizures. The ordinate represents pressure in millimetres of spinal fluid; the abscissa, time in minutes. The dotted portion of the curve denotes pressures obtained after the draining of fluid. The second half of the chart shows the pressure changes in the same patient when repunctured 24 hours later.

TABLE II. THE PERCENTAGE DISTRIBUTION OF THE PROTEIN CONTENT IN 793 CASES OF 'ESSENTIAL' EPILEPSY

Protein content (mg. per 100 c.cm.)	No. of cases	Percentage
7 to 19	98	12·3
20 to 45	612	77·2
46 to 85	80	10·2
85 to 100	2	0·2
100 to 200	1	0·1
	793	100·0

Colloidal Gold Reactions.—The colloidal gold reaction was determined in 741 cases. The method of Lange as described by Cockrill [12] was used. Any change in the gold solution greater than a lilac colour '2' was considered abnormal. None of our cases gave an abnormal colloidal gold curve and in only 60 cases (8 per cent.) was there a change as great as lilac '2' in the colloidal gold solution.

Wassermann Reaction.—Cases which received a diagnosis of syphilis of the central nervous system are excluded from this report. Convulsive seizures occur in about 30 to 60 per cent. of patients with general paresis [13] and about 17 per cent. of those with syphilitic meningitis.[14] The proportion is less in meningovascular syphilis and is hardly above the level of that of the general population in tabes dorsalis.

Sugar Content.—The sugar content of the cerebrospinal fluid was measured in 229 cases. The Folin-Wu method, modified by Rothberg and Evans,[15] was used. The results varied between 38 and 167 mg., with an average of 70 mg. per 100 c.cm. The percentage distribution of values is shown in Table III.

TABLE III. THE PERCENTAGE DISTRIBUTION OF THE CEREBROSPINAL FLUID SUGAR CONTENT IN 229 CASES OF 'ESSENTIAL' EPILEPSY

Sugar content (mg. per 100 c.cm.)	No. of cases	Percentage
38 to 50	7	3
50 to 60	35	15
60 to 80	154	67
80 to 100	24	11
100 to 167	9	4
	229	100

Eighty-two per cent. of the cases had values between 50 and 80 mg. per 100 c.cm. Of the 15 per cent. with values above 80 mg., some might be explained by the fact that in most instances the patients were not fasting at the time of the puncture. The absence of any very low concentrations speaks against hypoglycæmia as a significant finding in epilepsy.

Chloride Content.—The chloride content of the cerebrospinal fluid was determined in 209 cases. The Van Slyke [16] method or the Van Slyke method as modified by Wilson and Ball [17] was used and results expressed as milligrammes of sodium chloride. The greatest concentration was 794 mg. and the lowest 658 mg. per 100 c.cm. of fluid. The average was 723 mg. per 100 c.cm. The distribution of values is shown in Table IV.

TABLE IV. THE DISTRIBUTION OF THE CEREBROSPINAL FLUID CHLORIDE CONTENT IN 209 CASES OF 'ESSENTIAL' EPILEPSY

Chloride content (mg. per 100 c.cm.)	No. of cases	Percentage
658 to 675	2	1
675 to 700	9	4
700 to 760	194	93
760 to 794	4	2
	209	100

Only 3 per cent. of the values could be considered as definitely abnormal.

Calcium Content.—The calcium content was measured in 93 cases by the Clark [18] modification of the Kramer-Tisdall method. The values varied between 4·1 mg. and 5·8 mg., with an average of 5·0 mg. per 100 c.cm. Only seven cases had a value below 4·5 mg. and only one a value greater than 5·5 mg. per 100 c.cm.

Non-Protein Nitrogen.—The non-protein nitrogen content was determined in 10 cases by the Folin-Wu [19] method. The values varied between 11 and 26 with an average of 17 mg. per 100 c.cm.

SUMMARY OF INITIAL OBSERVATIONS.—The abnormalities in the cerebrospinal fluid in patients with epilepsy of undiagnosed ætiology were confined to a slight increase in pressure in 9 per cent., an increase of the cell count in 4 per cent., and a slight increase in protein content in about 10 per cent. of the cases. About one-fifth of the patients showed one or more abnormalities in the cerebrospinal fluid. Only six cases (one per cent.) had an increase in both pressure and the protein content.

Duration of Epilepsy and Cerebrospinal Fluid Abnormalities.—Tabulation was made of the cerebrospinal fluid pressures with relation to the age of the patient and also the duration of the history of seizures. The duration ranged from a few months to 45 years. The percentage of patients with

increased cerebrospinal fluid pressure was approximately the same for all age groups. Increased pressures were found in 8·3 per cent. of the 552 cases in which the duration of the disease was less than 10 years and in 10 per cent. of the 183 cases in which the duration was greater than 10 years. We therefore concluded that the age neither of the patient nor of the disease bears any significant relation to the pressure of the cerebrospinal fluid in these cases.

In tabulating the data with respect to any possible correlation between the increase in the protein content of the cerebrospinal fluid and age of the patient, we observed for each successive decade a slight progressive increase in the incidence of patients with an abnormal protein content. The proportion of patients with fluid abnormally high in protein content was 10 per cent. for those with a history of less than 10 years and 13 per cent. for those with a history longer than this.

Repeated Examinations.—This report deals only with the finding at the time of the patient's initial lumbar puncture at our hands. In comparison with other body fluids used for examination, the spinal fluid is relatively constant from one time to another. Even pressures, if made under standard conditions, are surprisingly alike. Examinations were repeated in many cases in order to check abnormal findings or apparent discrepancies. Checking was especially important in those cases with an abnormal pressure.

Of more interest are the examinations repeated at prolonged intervals. Twenty-eight patients had a second examination made from one year to as long as 11 years later. The number of cases in each of the first five years was successively five, six, two, four, and three. After seven years there were four, after 10 years one, and after 11 years two cases. The results of the second or third examination were with a few exceptions, as noted below, remarkably like the first.

Six of these 28 cases had a pressure of 200 mm. or greater at first puncture and all six had a normal pressure at the subsequent puncture one or more years later. In one case the pressure was 125 mm. at first puncture and 11 years later it was found to be increased to 220 mm.

The protein content was increased at first puncture in only one of the 28 patients. In this case already mentioned, a protein content of 200 mg. had fallen to 87 mg. after an interval of one year and to 56 mg. after 10 years. Two cases with original protein contents of 34 and 41 mg. had values of 47 and 52 mg. per 100 c.cm. respectively after a period of 11 years.

There was no significant change in the cell count or the colloidal gold reaction of any of the 28 cases.

SUMMARY

1. This report deals with the cerebrospinal fluid findings in 810 patients having seizures of undiagnosed origin.

2. The cerebrospinal fluid was entirely normal in approximately 80 per cent. of the cases.

3. An abnormal increase in pressure (between 200 and 300 mm.) occurred in 9 per cent. of the patients; only one per cent. had a pressure greater than 250 mm.

4. A slight pleocytosis (between 6 and 10 cells per c.mm.) occurred in about 4 per cent. of the cases.

5. The protein content was above normal in 10 per cent. of the cases; values greater than 85 mg. per 100 c.cm. were encountered in less than 0·5 per cent.

6. The colloidal gold and Wassermann reactions, the concentration of sugar, chloride, calcium and non-protein nitrogen content were normal in practically all cases.

CONCLUSIONS

The cerebrospinal fluid is normal in four-fifths of patients, the cause of whose seizures remains undetermined after clinical and laboratory examinations. A slight increase in pressure and in the protein content is the chief abnormality. Any marked abnormality in pressure or the contents of the fluid makes the diagnosis of 'essential' epilepsy hazardous.

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THE SENSATION OF VIBRATION, WITH SPECIAL REFERENCE TO ITS CLINICAL SIGNIFICANCE*

By

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The sensation of vibration is the subjective experience of an individual when a series of rapidly repeated stimuli are applied to certain of his sensory end-organs, probably those of touch, pressure and passive movement, the sensation being recognized by him as a continuous tremor. The sensation is evoked by applying a tuningfork to his skin, preferably just superficial to a bony prominence.

The study of the sensation of vibration, although it involves no instrument or technique that could not have been available for hundreds of years, has only been developed within the last fifty years, and even today little advantage is taken by the clinician of the exact and quantitive method of examination of the vibratory sensation which is now available.

BRIEF SUMMARY OF PREVIOUS WORK

Rumpf [29] was probably the first to make an exact study of the sensation. After a few preliminary experiments he used a series of 14 tuningforks with rates of vibration varying from 13 to 1,000 per second. He believed that the sensation was confined to the skin and transmitted by cutaneous nerves. Later Treitel,[34] using a fork of 128 vibrations per second, came to the conclusion that the sensation was distinct from pressure and touch, and that it arose in the soft tissues. He noticed the characteristic diminution of the sensation in tabes.

Gradenigo [14] developed a tuningfork, later much used in otological work, bearing the 'Gradenigo triangle.' A small triangle was impressed on the lateral side of the prong of a tuningfork, and appeared double, the one image overlapping the other, when the fork was struck. This overlap increased with diminution of amplitude of the prongs, and could be measured by a series of black lines, as seen under a magnifying glass. Egger [8] found that a tuningfork vibrating at a rate of 128 per second was the best felt, and that one of 2,048 per second was quite imperceptible. He called the sensation 'la sensibilité osseuse,' as he believed that it was perceived by end-organs in

* A résumé of a thesis accepted for the degree of M.D. of the University of Edinburgh.

bone, periosteum, ligaments and joint capsules. He noted decrease in the sensation with age.

Dweitschenko [7] noted that a thick layer of soft parts over the bone reduced sensibility. Rydel and Seiffer [30] came to the conclusion that the sensation was a function of deep sensibility and that all the superimposed tissues could take part in it. Sterling [31] stated that vibratory sensibility depended on the relative densities of the vibrating body and the tissues that carry the sensation.

Goldscheider [13] believed that vibratory sensation was not a special one, but the result of a peculiar stimulation of the end-organs responsible for pressure and touch. Knapp [19] found that a rate of 512 vibrations per second was the greatest rate normally perceptible. An instrument firm has recently been sending to medical men as an advertisement a small fork, of a vibration rate of 512 per second. Accordingly practitioners, if they bother to use the fork at all, will find the vibration sensation of many of their patients incorrectly reduced.

Minor,[24] using the Gradenigo triangle, stated, among some other important facts, that (*a*) the continuity of the bone was not necessary for a normal appreciation of the sensation; (*b*) periostitis, caries and superficial thickenings of bone have no effect on the sensation; (*c*) the vibrations were felt on bare bones and on sequestra; (*d*) the vibration sensation was diminished when the peripheral nerves were injured.

These facts suggested that while the bone had an important rôle in appreciation of the sensation it was not the site of its reception.

Williamson,[35] in a series of papers from 1905 to 1922, showed himself to be the foremost student of the sensation of vibration, with regard to its clinical application. He mentions its loss as one of the earliest signs of disease; its use in separating off pure diseases of the motor tracts; and that the tuningfork may be used in mapping out the upper limit of spinal lesions. He lists a number of diseases in which there is no loss, others in which diminution is an early sign. He did not, however, use a quantitative method of measuring the sensation. He mentions that the tuningfork is occasionally of advantage in detecting hysteria and malingering. In hemianæsthesia, if the fork is felt on one side of the sternum and not on the other, the disease is probably 'functional,' as the whole sternum is made to vibrate. Stated otherwise, in a case of anæsthesia of a limb, should the vibration not be felt when the fork is applied to a bone in the anæsthetic area, but be felt when applied to the same bone outside the anæsthetic area, the disease is likely to be hysterical. If the sensation of vibration is the only sensation to be diminished the disease is probably organic. In Rombergism the sensation is almost certain to be diminished if the disease is organic.

Bing [4] anæsthetized the skin with various substances, and found that vibratory sensation was slightly diminished, and distinguished it from that of touch.

Lewandowsky [20] and also Head [17] developed a method of testing the sensation which might be called the 'alternate displacement' or 'fatigue' method. The tuningfork is applied to a bone, say the right radius, and when the patient states that he can no longer feel the vibration a stop-watch is started and the tuningfork immediately transferred to the radius of the other arm, when the sensation will again be felt. The time is then taken until the sensation ceases to be felt in the left radius. The tuningfork is struck again and reapplied, first to the left radius, then to the right. Any marked difference between the two readings was held to be pathological. There are, however, serious objections to this method which may be dealt with here.

(*a*) The vibratory sensibility of the two sides of the body is often different, some writers stating that the sensation on the right, others that the sensation on the left, is the more acute.

(*b*) When one side is known to be abnormal the normal side cannot be measured against it, and has to be measured against a different limb, i.e. arm against leg. Usually the leg has a smaller sensibility than the arm.

(*c*) No account is taken of the force with which the fork is struck, i.e. its amplitude, for if the fork is started with a large amplitude its vibrations will last longer.

Experiment 1.—Strike the fork so that the amplitude is small, i.e. apply it just when the large window disappears (see later section for method) and test by Head's alternate displacement method, from right radius to left radius. Then do the experiment again, this time striking the fork until it clicks and applying immediately, i.e. when it is at its maximum amplitude. This can be repeated four times.

Result: (*a*) Fork at minimum amplitude, average of four trials 8·9 seconds (10, 7, 10, 8·5 secs.).

(*b*) Fork at maximum amplitude, average of four trials 11·5 seconds (11, 11, 13·5, 10·5 secs.).

That is to say, with Head's method the tuningfork is felt longer if struck harder. Head did not take account quantitatively of the strength with which the fork was struck.

The main conclusions from Head's [17] work on the nature of the sensation have been generally recognized. The tuningfork stimulates two aspects of sensation: (*a*) That of 'jarring contacts' which are appreciated by the thalamus (vibration is perceived, but differently, when the thalamus is cut off from the cortex); (*b*) That of rapidly repeated movements of small range. These are similar to the perception of passive movements, and are appreciated by the cortex.

Head emphasized the close correlation between the sensation of vibration and appreciation of measured movement and posture, and remarked on the similarity of their tracks in the cord. As no lesion above the thalamus can cause complete insensibility to vibration, except in neural shock, the test of the tuningfork is only useful as a sign of the integrity of the posterior columns. He points out that developmentally, in the lateral line of fish, sensations of posture and vibration are associated, and both sensations have

a temporal and spatial factor, the former stressed most in vibration, the latter in posture.

Symns [32] developed what is undoubtedly the most useful advance in the use of the tuningfork. He used a large A fork with a vibration rate of 108·75 per second (supplied by Messrs. Down Bros.). The feature of this fork is the presence between the prong tips of three interlapping plates of metal (see fig. 1). One of these plates, which fits in between the two on the other prong, bears two notches, which are invisible when the fork is at rest, but visible when it is struck fairly hard. The fork is struck and applied to the body just when the notch, or window, is about to disappear, and a stop-watch is started at the same time. When the patient states that he can no longer feel the sensation the watch is stopped and the time taken. This method ensures that the fork is always applied at a given amplitude, and results are quantitatively comparable with results from other parts of the

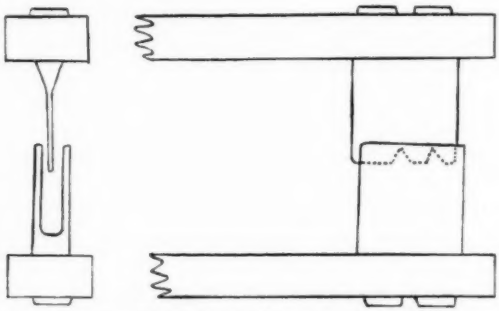

FIG. 1.—Head of tuningfork as used by Symns.

body and results from other individuals. Unfortunately results are not necessarily comparable with different forks, for every worker since Symns, using a presumable identical fork made by the same firm, has agreed that Symns' results are far too high. The firm investigated that matter, and found that the discrepancy was probably due to differences in temper and weight of the prongs. To avoid this in future, all forks made by that firm are tested against a known normal member of its staff and its graph is delivered with every new fork. The results can be plotted on a graph.

Symns dealt with the results in diabetes, tabes and disseminated sclerosis (discussed farther on). In alcoholic neuritis the legs and arms were always affected, the sacrum sometimes. In plumbic neuritis the legs were commonly, the arms less commonly, involved. There was no loss in amyotrophic lateral sclerosis, paralysis agitans and progressive muscular atrophy. Among other general statements he declared that: (*a*) when vibration sensation over the sacrum is absent the cord is involved; (*b*) when present over the sacrum but absent over one other extremity the condition is a peripheral neuritis.

Symns believed that the stimulus is carried through the skin and soft parts, and transmitted along the bones, but is felt in the soft tissues. A small tuningfork may not be felt over anæsthetized skin, when a larger one will be, i.e. both light touch and deep pressure are involved.

Wood, [37] using 100 normal cases, established a limit of normality, which cases are the basis of the published charts. He found the same sacral dip in tabetics that was emphasized by Symns, and remarked on the frequency with which patients, being treated as cases of gastric and duodenal ulcers, were later shown to be tabetics with gastric crises, as a result of routine

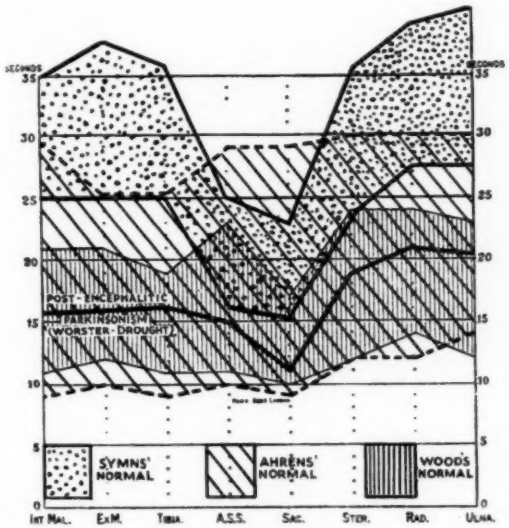

FIG. 2.

testing with the tuningfork, followed up by examination of the cerebrospinal fluid.

Piercy [27] followed up the work of Symns and Wood, and agreed with them in the main. In most cases of syphilis confined to the upper motor neurone no marked changes could be found, as would be expected, the vibratory test being a sign of integrity of the posterior columns. He remarked also on the changes in old age and chronic alcoholism.

Ahrens [2] also worked out a series of 100 normal cases, and found the spread of his graphs to be slightly wider than those of Wood's (fig 2). He emphasized the influence of the intelligence of the patient, his state of mind, fatigue and extraneous distractions. He worked out the graphs of a series of cases of pernicious anæmia, diabetes and general paralysis. Most of the latter had tabetic curves, in contradistinction to the findings of Piercy.[27]

Epstein [9] describes the route in the cord and brain that the nervous impulse takes. The neurones of the first order travel in the columns of Goll and Burdach and end in the nuclei gracilis and cuneatus. Neurones of the second order relayed from these nuclei decussate at the median lemniscus, and end in the optic thalamus, which is the subcortical station subserving primitive undifferentiated sensation. Thalamocortical paths of neurones

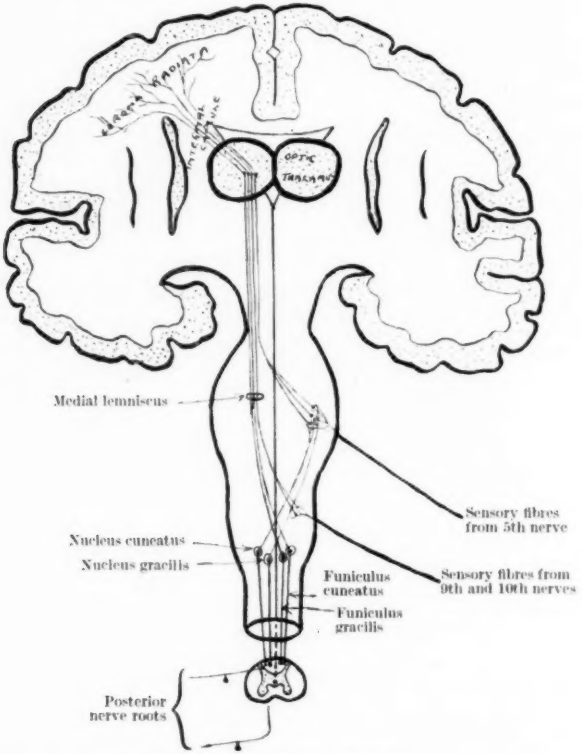

FIG. 3.—Adapted from Epstein.

(third order) traverse the posterior end of the posterior limb of the internal capsule, pass into the corona radiata and end in the sensory cortex, which organizes and analyses the perceptions (fig 3).

Epstein emphasizes the value of the fork in multiple neuritis due to lead, arsenic, etc., its use in hysteria and in diagnosing pure affections of the motor systems, such as progressive muscular atrophy.

McKinley [22] mentions the essential physiological error in the previous quantitive methods of testing, as developed by Symns. The latter applied

the fork at its maximum amplitude, which gradually decreased to a minimum when the patient ceased to feel the vibrations; the scientific method would be to begin from below and find the threshold. Also fatigue brings a big error into Symns' method. To show this I have devised a simple experiment.

Experiment 2.—The fork was applied to the right radius when vibrating at a known amplitude. It was felt for 23 seconds. It was reapplied vibrating at the same amplitude, but every five seconds was held away from the bone for five seconds' rest. Its vibrations were felt up to the 32nd second.

This shows that the patient ceases to feel the vibration long before the amplitude of the fork has fallen to its physiological threshold, his senses being dulled by fatigue; so we have been measuring two factors—threshold of amplitude, and fatigue.

To obviate this McKinley developed a method of applying the fork at

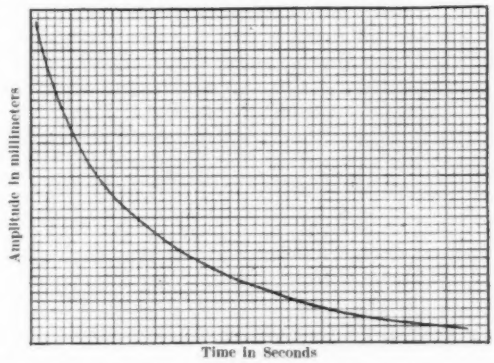

FIG. 4.—Schematic graph (after McKinley) to show relation between time and amplitude.

its minimum amplitude. Between the prong tips could be applied one of a series of rods of which he had 15, of differing lengths. The rod was withdrawn sharply and the fork vibrated at an amplitude depending on the length of the rod. This method is much inferior to the electrical one of Gray [15] and not nearly so valuable clinically as the method of Symns. The graph of time plotted against amplitude shows that the fork runs down very rapidly at first (fig. 4).

Pearson [26] mentions the neglect of the age factor by most other authors, and discusses the diminution of the sensation decade by decade. To explain this he describes some early work by Williamson [35 (h)] on the vessels of the cord. The vessels of the cord arise from spinal arteries that are among the longest vessels in the body. In the lumbar cord they are less numerous than in other portions, and must pass upwards. Arterial changes are thus common in these vessels in old people, giving rise to degeneration of the

posterior columns, hence to changes in the sensation of vibration, more marked in the lower limbs.

Gray,[15] reasoning from the same objections as McKinley, has developed the most accurate method of testing the sensation of vibration. A magnetic coil is set between the prongs of the fork and the desired amplitude set up by varying the amperage of current running through the apparatus. The fork is not struck, but its vibrations sustained electrically. Obviously the greater the amplitude of the threshold stimulus, the less is the acuity of the sensation. The lowest constant amplitude felt by normal persons was 2·34 mm.

His work was confined to normal cases and to cases of pernicious anæmia. He noted the loss of acuity in pernicious anæmia, and went into great detail in its distribution. He also noted the loss in old age. An interesting feature he revealed was that the threshold improved after repeated testing. The threshold value at one point on September 12, 1929, was 0·22 mm., and on the same point seven months later 0·018 mm. On another point it was 0·549 mm. and 0·031 mm. respectively on the same two dates.

My criticism of Gray's method is that although it is scientifically accurate it is (*a*) far too cumbersome and lengthy for clinical use; (*b*) his thresholds for one point have a much larger spread than the equivalent threshold-fatigue-time method of Symns; (*c*) his method shows nothing clinically that cannot be shown by the Symns' fork; (*d*) if it is insisted that the threshold be approached from below and not from above, as in Symns' method, and the element of fatigue be eliminated, there can be devised a method of using the Symns fork that fulfils these criteria, and is no more involved.

AN ORIGINAL METHOD OF TESTING THE SENSATION OF VIBRATION

The ideal method of testing the sensation of vibration should (*a*) approach the threshold from below, (*b*) give the result in millimetres of amplitude of the fork, (*c*) not be so cumbersome that it cannot be used in routine examinations, and (*d*) eliminate the factor of fatigue.

Symns' method does not fulfil (*a*), (*b*), and (*d*); Gray's and McKinley's method, (*c*).

If the fork as used by Symns be struck and the stop-watch started at a known amplitude, i.e. when the first window disappears, the amplitude decreases, with a decreasing rate which can be plotted against the time (fig. 4). The fork is kept vibrating for, say, 30 seconds, and then, and not until then, applied to the bone. The patient is asked to state whether he can feel the vibrations or not. If he can, the process is repeated, increasing the length of time that the fork is held away from the bone until a time is reached when the vibrations cannot be felt. The previous reading, expressed in seconds, is the threshold value for that bone. Similarly, if at first the vibrations cannot be felt, the time is reduced until they can be. In practice, as the amplitude decreases with time, it is not necessary to change the latter

units into the former, especially as a complicated apparatus is required to plot the required graph. Three, and not more than four, applications are necessary to find each threshold, and they may be reduced to two. In the present instance the time-intervals were increased or decreased by 2·5 seconds. Two graphs of a normal individual are compared (fig. 5).

The continuous line is the threshold-fatigue curve of Symns, the dotted line is the minimum-threshold method, as it might be termed. A striking parallel is seen between the two curves, which speaks much for the accuracy of both methods. A disadvantage, which is easily rectified, is the length of time one has to wait for each application, i.e. 25 to 40 seconds. This is because the fork is made to be applied at a relatively high amplitude. If it

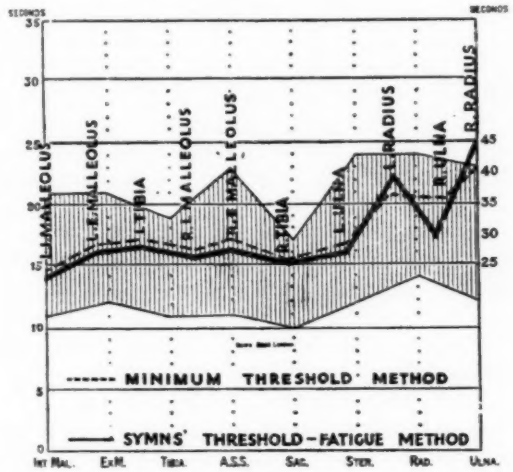

FIG. 5.

were applied at a lower amplitude the delay would be much less. To lower the amplitude it would only be necessary to have a larger window made in the metal slot.

The great advantage of the method is the clear-cut 'yes or no' reply required from the patient, who either feels the vibration or does not—very different from the hesitant replies often obtained from the most intelligent patients by the method of Symns, when he or she has to say when a slowly disappearing sensation becomes imperceptible.

In the case of, say, the right radius, the vibrations are only felt for 25 seconds, starting from a given amplitude, if the fork is continuously kept applied to the bone. The true threshold value for the sensation is, however, only reached after 40 seconds. The difference, 15 seconds, is loss due to fatigue, and perhaps slightly to an inability to appreciate a correct end point when the threshold is approached from above.

THE NATURE OF THE SENSATION

There is unanimity among observers as to the route taken by the nervous impulse in the nervous system, and this was described before. Disagreement arises in the study of the site of reception of the stimulus.

Rumpf [29] believed that the sensation was confined to the skin. Treitel [34] thought that it was distinct from both pressure and touch, as opposed to Goldscheider,[13] who maintained this relationship. Egger [8] was the first to put forward the idea that the end-organs of the sensation lay in bone, periosteum, ligaments and capsules of bones and joints. Minor [24] in a reasoned paper showed that continuity of bone was not necessary for perfect reception of the stimulus; that periostitis, caries and superficial thickenings of the bone had little effect, and that the sensation could be felt on a dead sequestrum. All this pointed to the fact that the bone was probably not the site of reception. Bing [4] differentiated the sensation from that of touch by excluding the latter with local anæsthesia. Head [17] analysed the subjective sensation into the two factors of 'jarring contacts' appreciated by the thalamus, and rapidly repeated movements of small range, perceived by the cortex. He noted the relationship between the sensations of vibration and those of passive movement and posture. Symns [32] agrees. Tilney,[33] Epstein [9] and Gray [15] believe that the sensation arises in bone.

A few facts should be noted. (*a*) The sensations from a small tuning-fork of 518 vibrations per second may be abolished by local anæsthesia, and not those of a larger fork. (*b*) Disease and injury of bone, however, have very little effect on the distinctness and duration of the sensation. Three cases were investigated, and may be cited. In one, the affected femur was the site of extensive chondromatosis. In another there was decalcification with a suggestion of fibrocystic disease, and in the third the affected limb, due to disease-atrophy, was considerably shorter than the other. In each case the sensation in the affected limb was as good as in the normal, except in the third case where it was better.

> *Experiment* 3.—The vibratory sensation was estimated on the skin of the posterior aspect of the middle of the thigh, and found to be 7 seconds. A plaster cast about 7 inches wide was then made to fit around the middle of the thigh, and the fork applied to it after it had set. The sensibility was slightly increased to 11 seconds. The fork was next applied to the condyles of the femur, and the time of reception of the stimulus was found to be still longer, i.e. 17 seconds.

Thus the bone, as a sounding-board in the middle of the soft tissues of the thigh, gives better results than the plaster-cast, a sounding-board applied to the tactile organs of the skin.

The sum of the evidence points to the fact that bone, because of its structure, distributes its vibrations to the deep tissues, i.e. muscles, tendons, joints, fascial planes, perhaps periosteum, where the main part of the sensation is perceived. The skin, to a less extent, takes part in its reception.

SOME INTERESTING FEATURES

Fatigue.—The sensation of vibration can be dulled by fatigue. Of this there are two forms, which may be termed intrinsic and extrinsic fatigue, a distinction apparently not hitherto drawn. Intrinsic fatigue is the diminution of the acuity at the point tested due to the continued application of the fork. This has been discussed earlier. Extrinsic fatigue is due to fatigue of the organism as a whole.

Experiment 4.—The sensation was tested in an individual before and after a round of 18 holes of golf.

	Before	After
Right radius	22 seconds	16 seconds
Right ulna	22 ,,	16 ,,
Left radius	22 ,,	17 ,,
Left ulna	19 ,,	16 ,,

These results show a considerable diminution in the sensation.

Vocal Fremitus.—Various workers have remarked on the difference in vibratory acuity of the two sides of the body. Some have stated the sensation on the left side of the body to be the more acute, others, that on the right. In this study the difference was noted, but no special side was found to predominate. This proves the clinical importance of testing vocal fremitus with the same hand on each side of the chest.

CLINICAL APPLICATIONS

A series of approximately 100 cases was examined for this work. They include 25 cases of diabetes and 25 cases of pernicious anæmia. Although the numbers are rather small for statistical analysis, the results may be considered suggestive.

The technique adopted was that developed by Symns.[32] The fork used is a large A fork, of 108·5 vibrations per second, and prongs just under 9½ in. long. Each fork supplied is tested against a normal individual in the employ of the manufacturers, and his graph delivered with the fork. The fork is struck, and when the first window just disappears a stop-watch is started, and the handle of the fork applied to the point to be examined. The patient should be at ease, not tired, and should, before beginning the real test, be made to understand just what he has to do. During the test the patient can be examined as to his reliability by applying the fork when the vibrations have been stopped. When the patient ceases to feel the sensation he says 'Yes,' and the watch is stopped. With a reasonably intelligent patient successive tests show considerable accuracy, and need not vary more than 10 per cent. on repeated testing. The points tested are the radius, ulna, sternum, sacrum, anterior superior spine, tibia, internal and external malleolus. Both sides are tested, and if there is any appreciable difference the two results are recorded on the same graph. The fork should be applied with

a constant pressure, firm, but not heavy, care being taken not to hurt the patient. Leading questions will of course be avoided.

Ten cases not suffering from disease of the nervous system were tested, not to set a standard of normality, but to check the tuningfork with the comprehensive series of normals already investigated by Wood [37] and Ahrens.[2] The spread of the curves is certainly wider than that which Wood described as normal, but except for one case, fits in with that described as normal by Ahrens. This exception, in which the diminution of sensibility was slight, was a neurasthenic.

The compound graph (fig. 6) shows clearly a feature that has been described by others, the tendency for the vibration sensation in the arms to

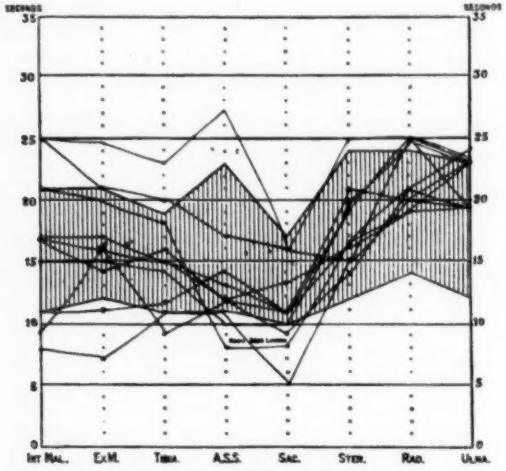

FIG. 6.—Normal cases (10).

be fairly high, and to approximate closely in different individuals, whereas the spread over the limbs is much wider.

Loss with Age.—The gradual loss of the sensation of vibration has been remarked upon by Egger,[8] Rydel and Seiffer,[30] Piercy,[27] Pearson [26] and Gray.[15] Pearson made the most complete study of it, and stated that in the legs there was a diminution decade by decade, but no change in the arms occurred until the fiftieth year. Gray noted the effect of age on the vertebræ and pelvis, where there was also a loss. The five cases presented (fig. 7) include two with fracture of the neck of the femur, one with cataract, one with eczema, one with cholecystitis. The ages varied from 71 to 84 years. One had a low normal graph, all the others were distinctly abnormal. The loss was in the pelvis and lower limbs, the sensation in the arms remaining normal but on the low side. The loss, as explained before, is most likely the result of arterial degeneration of the spinal vessels.

Sensation in the Blind.—There does not appear to have been any work done on the sensation of vibration in the blind. It is often loosely stated

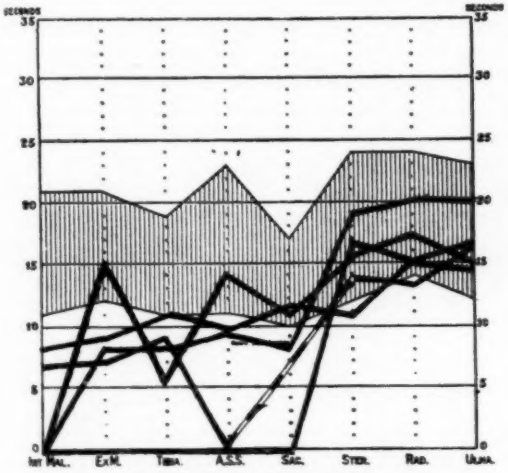

FIG. 7.—Old age (5 cases).

that the sensitivity of blind individuals is increased, without either adequate quantitative proof or evidence that the apparent increase is not due to a

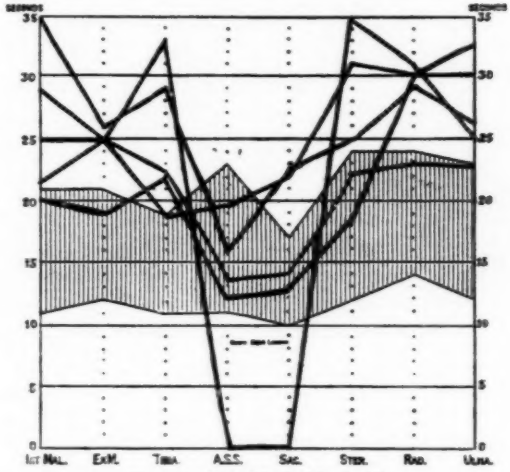

FIG. 8.—Blind individuals (5).

raising of the threshold for the sensation but to a better discrimination of what is felt. The sensation of vibration is a useful test for this problem,

as it can be quantitatively measured, and it is unlikely that the individual tested can have had much previous experience of the sensation, resulting in better discrimination.

Results show a considerable increase in the vibratory acuity in the blind. Of the five cases (fig. 8), two have high normal curves, two have curves distinctly above the normal, and the fifth has very high acuity in the limbs, but practically absent vibratory sensibility in the pelvis. On the whole in the five cases the increase tends to be in the limbs and not in the pelvis. There is very little difference between the upper and lower extremities.

It should be noted that all the individuals selected were between the ages of 21 and 41, healthy, and had been blind for more than 10 years, and

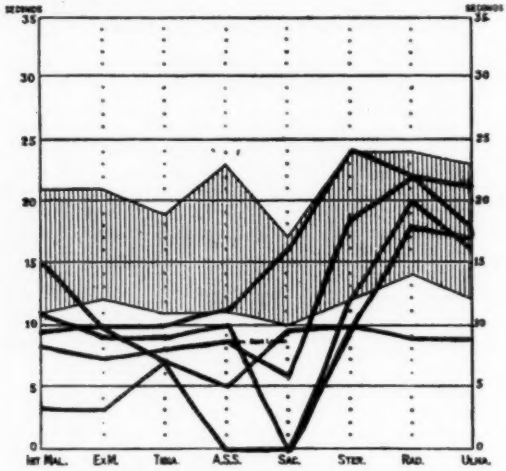

FIG. 9.—Syphilitic aortitis (5 cases).

were trained workers. No cases were included in which the blindness might have been due to lesions of the central nervous system. Gray [15] notes that the normal individual in whom he tested the sensation of vibration over a period of months showed a marked increase of acuity. Apparently the training of the apparatus of sensation which takes place in the blind over a period of years increases the threshold for sensation both generally, and for specific sensations.

Syphilitic Aortitis.—This series of cases was examined to test an assertion by Symns [32] that absence of the sensation of vibration at the sacrum denoted a lesion of the central nervous system, the lesion in this case being easily demonstrated by the examination of the cerebrospinal fluid. None of the patients showed obvious signs of a lesion of the central nervous system.

The first impression from the compound graph (fig. 9) is a lessening of the sensation in the pelvis and lower limbs. This general diminution is

probably due to the average age of the group, which is 56. The only patient below the age of 52 had the only normal curve. Two cases had absent vibratory sensation at the sacrum. The cerebrospinal fluid of one was normal, that of the other showed the following: W.B.C.—50 per c.mm.; globulin—excess; albumin—0·05 per cent.; Fehling's solution—reduced; colloidal gold test—1112100000.

This shows that absence of sensation of vibration at the sacrum cannot be taken as diagnostic of cord involvement, but is suggestive, and might be used as a method of weeding out patients. Piercy [27] suggests that absence of sensation of vibration at the sacrum in a patient in whom no cord lesion can be found is suggestive of a healed lesion. This would be difficult to prove.

Postencephalitic Parkinsonism.—In this condition both Williamson [35]

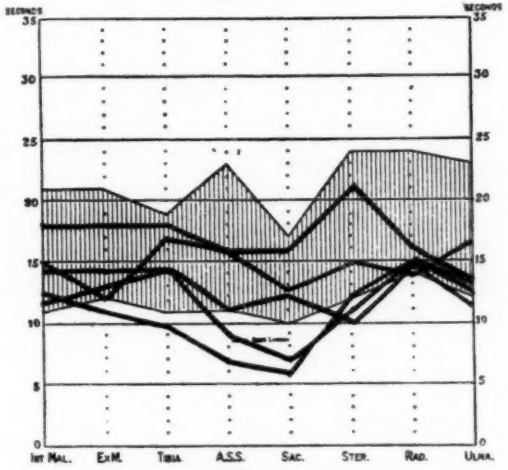

FIG. 10.—Postencephalitic Parkinsonism (5 cases).

and Symns [32] state that there is no change in the acuity of the sensation. This is of course the accepted view, as no change in sensation is usually described.

Worster-Drought and Hill,[38] however, come to a different conclusion, stating that there is a loss, worse with the severer forms of the disease, and on the more affected side. It appears, however, that although they used Symns' technique, they also accepted what Symns described as the normal. Since it has since been amply proven that Symns' results were too high, due to the mechanical features of the fork he used, their conclusions cannot be accepted. They give the mean graph from their cases in their paper, and it is seen that this falls entirely within the normal as described by Wood and Ahrens (see fig. 2).

The compound graph (fig. 10) of the case sdone for this work demonstrates

that the curves are mostly in the low normal zone. The average age for the group is 40. The feature of each individual curve, as seen in this group, is its evenness, i.e. the readings at different parts of the body do not vary greatly from one another.

Tabes.—Treitel [34] originally described a loss of vibratory sensibility in tabes. Williamson [35] stresses its early appearance. Symns [32] demonstrated its typical graph when tested with his technique. He stated that there was always loss over the sacrum, and usually over the lower limbs, occasionally in the upper limbs, and that there was a correlation between loss of vibratory sensibility and Rombergism. Wood,[37] Ahrens,[2] Piercy [27] and Epstein [9] all agree with this. The five cases presented here are all those

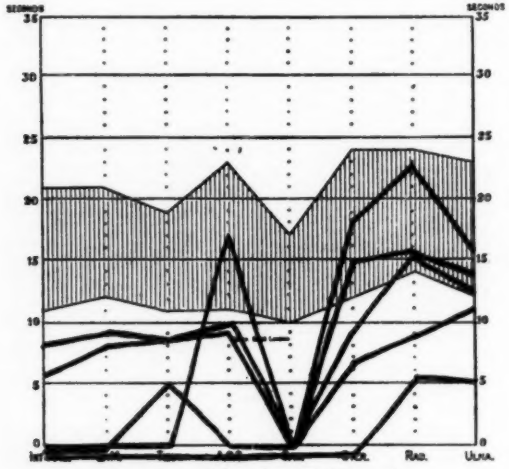

FIG. 11.—Tabes dorsalis (5 cases).

of typical chronic tabetics, who had been attending a venereal diseases' clinic. All the graphs (fig. 11) are typical, and show the 'sacral dip.' In two, the sensation was diminished in the upper extremities. One patient has the lowest sensibility of anyone I have yet tested, having anæsthesia to vibration at all points tested, except the left arm. It must be emphasized that the typical graph is not diagnostic, but suggestive.

Disseminated Sclerosis.—In this disease Williamson [35] stated that loss of vibratory sensibility was often the only sign of the disease on the sensory side. Symns [32] stated that there was always a loss over the sacrum, and sometimes over the long bones. In this series (fig. 12) all had loss over the sacrum, and loss or diminution in the limbs, except for one case, age 29, the youngest. In view of the varying clinical pictures the disease presents it would be natural to find inconclusive findings in so small a series. However, interest lies in the fact that one sensory change can nearly

always be found in this disease where sensory disturbances are often hard to demonstrate.

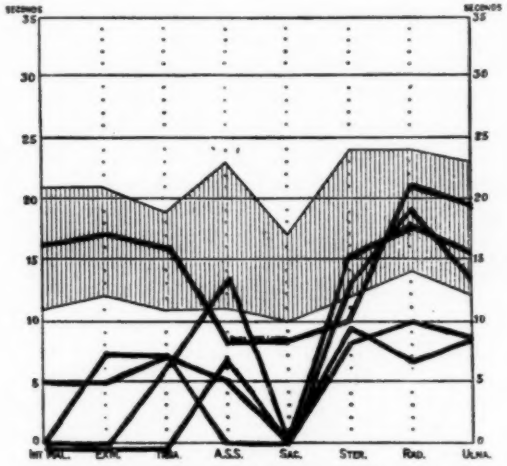

FIG. 12.—Disseminated sclerosis (5 cases).

Microcytic Anæmia.—This series of cases (fig. 13) is presented as a contrast to pernicious anæmia. The average age is somewhat high, 55 years.

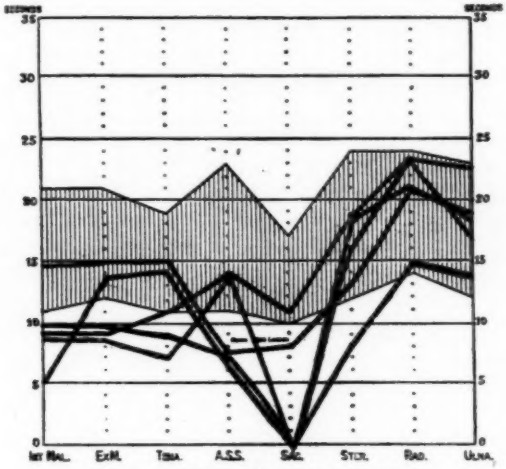

FIG. 13.—Microcytic anæmia (5 cases).

and a lowering of the curves must be expected. Two of the patients have normal curves for their ages. Three have absence of vibratory appreciation at the sacrum, but only one a diminution over the lower limbs. According

to Symns [32] these three should have cord lesions. One had other signs and symptoms suggestive of subacute combined sclerosis, the complication from which the patient would most likely have been suffering, but it was hardly likely that the other two could have been suffering from a nervous complication. At least one shows a vibration chart suggestive of tabes.

Diabetes.—Williamson,[35] Symns,[32] Ahrens [2] and others describe loss of the sensation of vibration in diabetes. Williamson states that the loss is often so slight that it can only be demonstrated with the tuningfork and that it is often worse on the side affected by gangrene or ulcers. Ahrens

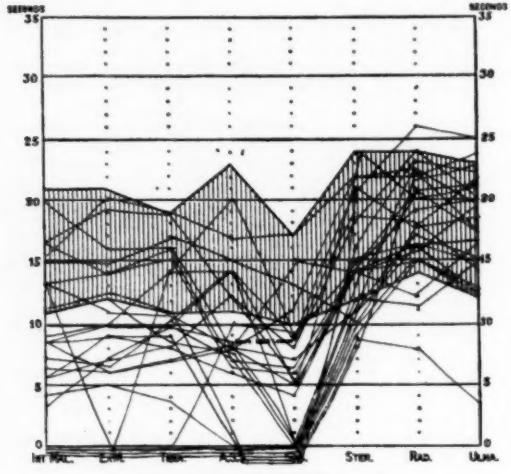

FIG. 14.—Diabetes (25 cases).

agrees. I have not found that the presence of arterial changes in the limbs usually causes diminution on the affected side.

The following table shows the incidence of absent or deficient sensation of vibration in the 25 cases :—

	Sensation absent	Sensation deficient	Sensation absent or deficient
Ulna	1	0	1
Radius	1	0	1
Sternum	0	5	5
Sacrum	9	5	14
Ant. sup. spine	5	2	7
Tibia	4	4	8
Ext. malleolus	5	5	10
Int. malleolus	4	5	9

It will be noted (fig. 14) there is derangement at the sacrum in 14 cases, or 64 per cent. Sensation was never absent at the sternum. In the 55 cases of derangement at the points tested, 26 showed only diminution and not loss. It is important to note that without some quantitative technique this would probably not have been noted.

According to Symns' theory two of the cases are pure peripheral neuritis, without cord involvement, and five cases show cord involvement without peripheral neuritis. This is hardly likely to be the true state of affairs, and it cannot be granted that absence of sensation of vibration at the sacrum is a sign of a cord lesion. Five further cases show a deficiency both in the limbs and in the pelvis. Twelve of the 25 cases, or 48 per cent., show changes that definitely point to a lesion of the nervous system, probably a peripheral neuritis, with a possibility of one or two cases having a cord lesion, probably subacute combined sclerosis.

Pernicious Anæmia.—The history of the description of nervous lesions in pernicious anæmia is interesting. Addison [1] did not describe lesions of the central nervous system in his original account of the disease. Biermer [3] described weakness, giddiness and palpitation, and found capillary hæmorrhages in the brain. In the early days subacute combined degeneration of the cord was confused with tabes. Dejerine [6] pointed out the loss of deep sensibility in subacute combined degeneration, and its relation to pernicious anæmia. He also described the symptoms as due to lesions of the long fibres in the columns of Goll and Burdach.

Woltmann [36] suggested that some of the nervous lesions might be the result of a peripheral neuritis. Hamilton and Nixon [16] carried out this idea still further, and believed that the remissions in the nervous symptoms might be due to the healing of the neuritis, and not to cord involvement. They based their conclusions largely on pathological data. Bramwell,[5] on the other hand, stated that absence of lesions of the peripheral nerves was a feature of the disease.

Hamilton and Nixon [16] found that loss of vibration sense was the commonest sensory deficiency found in subacute degeneration of the cord, being commoner than loss of the sensations of position, touch, or superficial pain.

Various workers have estimated the percentage of lesions of the central nervous system in pernicious anæmia. Some estimates are as follows :— Bramwell [5] 2·8 per cent. ; Nonne [25] 11·7 per cent. ; McCrae [21] 25 per cent. ; McPhedran [23] 40·9 per cent. ; Henneberg [18] 50 per cent. ; Gray [15] 66·6 per cent. ; Hamilton and Nixon [16] 80 per cent. ; Woltmann [36] 80·6 per cent.

Symns,[32] Williamson [35] and Friedman [12] emphasize the use of the tuning-fork in subacute combined degeneration, the latter considering it to be one of the most efficient tests. Gray [15] made a fairly complete examination of a series of pernicious anæmia cases with his special tuningfork, and found that the minimum amplitude felt showed an increase in pernicious anæmia

which was not sufficiently large to be diagnostic in the arms, but was so in the lower limbs.

The following table embodies the results of the examination of 25 cases of pernicious anæmia :—

	Sensation absent	Sensation diminished	Sensation absent or diminished
Ulna	0	6	6
Radius	2	3	5
Sternum	0	6	6
Sacrum	14	4	18
Ant. sup. spine	11	6	17
Tibia	7	8	15
Ext. malleolus	12	5	17
Int. malleolus	12	6	18

In no case (fig. 15) was the sensation absent at the sternum or ulna. At 102 points tested there was derangement of sensation, but diminution

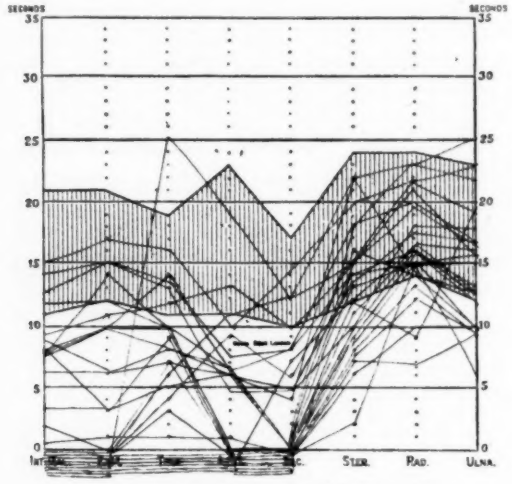

FIG. 15.—Pernicious anæmia (25 cases).

only in 44 of them. There was derangement at the sacrum in 18 cases, or 72 per cent.

According to Symns' theory there are in my group four cases of pure peripheral neuritis, four cases of pure cord lesions, and ten cases where the lesion lies in both the cord and peripheral nerves. In view of the results discussed in diabetes it is difficult to accept this theory.

Eighteen of the series are shown by the tuningfork to have definite lesions of the nervous system. This is 72 per cent., and the percentage would doubtless be higher were other criteria used as well as the sense of vibration as a sign of nerve lesions, and would doubtless approach that given by Hamilton and Nixon, and Woltmann, of 80 per cent. Ahrens [2] stated that none of his cases had normal sensation of vibration. In this series four were normal and three doubtful. The average age of the normal cases was 40 years, and of the whole group 54 years.

Miscellaneous Cases.—A series of cases are shown to illustrate special points of interest, or to demonstrate the value of the use of the tuningfork, or even, occasionally, the added confusion to which its use might give rise.

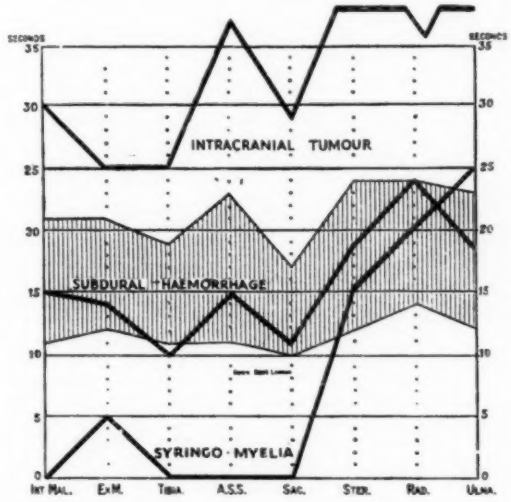

FIG. 16.

Subdural Hæmorrhage.—A case, the result of a fall, was seen. It was diagnosed after lumbar puncture, adhesions having resulted, causing a block in the flow of cerebrospinal fluid. This gave rise to Froin's syndrome; lipiodol, by cisternal puncture, was held up above the adhesions. The vibration sense was normal (fig. 16), showing that there could not have been much damage to the posterior roots or columns, although the patient had a paresis with absence of reflexes in the lower limbs. This was proved by the clinical course of the disease, the patient making a perfect recovery with no surgical interference.

Here may be mentioned some conflicting views in the literature, Williamson [35] stating that there is no loss in cord tumours and hæmatomyelia, Epstein [9] stating that there often is. On purely anatomical grounds it is obvious that the site and extent of the lesion will be the deciding factor.

Intracranial Tumour.—An interesting but isolated case of increased intracranial pressure, probably due to a tumour, is presented. The site was not definitely diagnosed, but a suboccipital decompression gave much relief. The interest lies in the marked sensory acuity of the patient, the highest that I have yet tested, the readings being in some places too high to plot on the graph. In addition, the patient was markedly sensitive to pain of all kinds, intravenous injections causing him much discomfort. Further speculation on one isolated case would be useless. A similar result was not obtainable in another case of intracranial neoplasm.

Syringomyelia.—Williamson [35] states that there is no change in the vibration sense in syringomyelia, and Epstein [9] that there might be. The

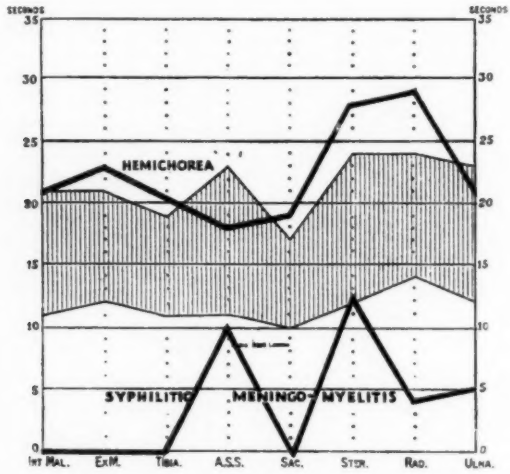

FIG. 17.

only case in this series showed marked loss of the sensation in the lower extremities (fig. 16).

Hemichorea.—To illustrate the absence of changes of the sensation of vibration in lesions of the brain, a case of hemichorea, probably the result of encephalitis lethargica, is presented. The patient had choreiform movements confined to the left side of the body. In view of the sites known to be affected in both encephalitis lethargica and lesions giving rise to chorea, i.e. the basal ganglia and midbrain, it is interesting that the sensation of vibration should have escaped entirely (fig. 17).

Cerebrospinal Syphilis.—According to Piercy [27] lesions of the secondary neurones should not give rise to diminution of vibratory acuity, hence in a pure case of general paralysis there should be no change, while there is a loss in tabes. Ahrens [2] found that of his five cases of general paralysis

three had normal graphs and two tabetic ones. It is difficult to be dogmatic as so many cases are a mixture of the two. A case of cerebrospinal syphilis may give a graph suggestive of tabes, but the patient may be suffering from syphilitic meningomyelitis, as in the example presented here (fig. 17).

Amyotrophic Lateral Sclerosis.—This disease, being purely a lesion of the motor neurones, should display no diminution of the sensation of vibration. In one of the illustrative cases (*a*) this was the case, but the other (*b*) presents an interesting problem. This patient had all the signs and symptoms of a pure upper motor neurone lesion, but absent vibratory sensation at the sacrum and anterior superior spines. Should this be sufficient to change the diagnosis? In this case I believe not, as the individual was rather fat,

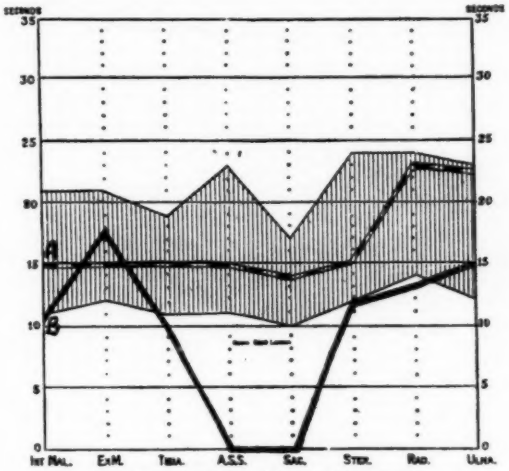

FIG. 18.—Amyotrophic lateral sclerosis.

and had thickened arteries, which if present in the lumbar cord would suffice to cause this deficiency.

Peripheral Neuritis.—All writers concur that vibratory sensation is diminished in peripheral neuritis. Symns [32] states that the sensation is usually present in the sacrum, but absent in one or other of the extremities. In diabetic neuritis he declared that the arms were normal, but the legs affected. In alcoholic neuritis both arms and legs were affected, and in plumbic neuritis usually the legs and less so the arms. Of my 25 cases of diabetes, only one had diminution in the arms, but 15 in the legs.

Two cases of peripheral neuritis, both of unknown origin, one acute, are presented here. Both show typical curves (fig. 19), the sensation being present at the sacrum, but absent at the limbs. In both of them the tuning-fork was of help in coming to a diagnosis. In this disease the sensation of vibration is usually the first to disappear, and is often deficient before the

kneejerks are absent. This is well shown in case (*a*). The kneejerk was present on the left side, but the tuningfork, though felt in the middle of the

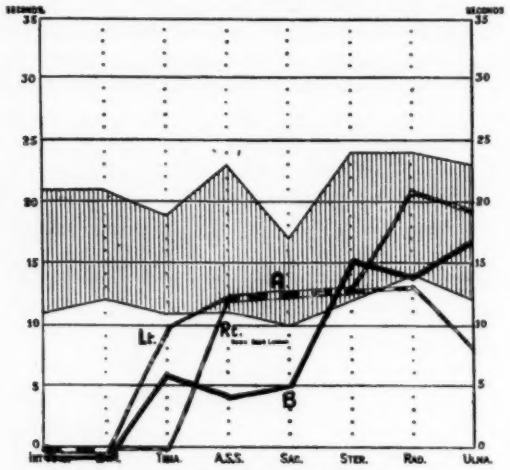

FIG. 19.—Peripheral neuritis.

shaft of the tibia, could not be felt at the malleoli. The kneejerk was absent on the right side, and the sensation of vibration lost in the whole tibia, as well as at the malleoli.

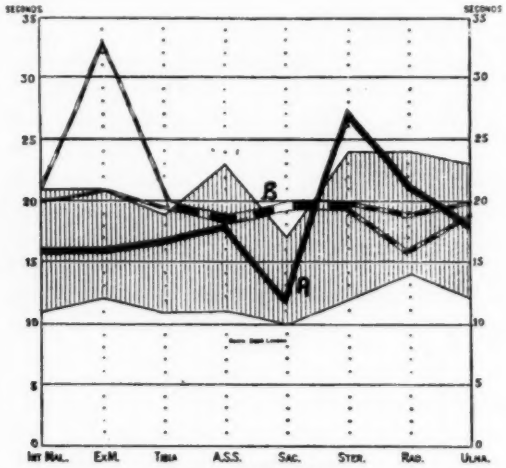

FIG. 20.—Hemiplegia.

Hemiplegia.—Head,[17] Epstein [9] and others have discussed the effect of cerebral lesions on the sensation of vibration. Usually there is little loss,

but if there is other sensory diminution there is often some deficiency of the sensation of vibration. It is never completely lost, however, except in cases of neural shock. The protopathic component of the sensation, or what Head calls the 'jarring,' is probably felt in the thalamus, and the epicritic in the cortex. In hæmorrhage in the internal capsule there is said often to be a diminution, or feeling of distance, in the sensation. On the other hand, when the thalamus is irritated, or divorced from the cortex, the sensation is often extremely unpleasant, or even painful, and may be felt more strongly than in the sound limb. This applies to other sensations as well as vibration, such as tickling or pin-pricking, and was described by Roussy [28] under the name of 'syndrome thalamique.'

Of the two cases of hemiplegia presented (fig. 20), both aspects are involved. In case (*a*) the sensation in the paresed limb was felt as long as in the normal limb, in spite of the fact that the patient averred that the sensation was much weaker on the paralysed side. The other patient (*b*) showed clearly the 'syndrome thalamique,' as when the fork was applied to the external malleolus of the affected side he cried out with discomfort, and attempted to withdraw his foot. The sensation was felt there much longer than in the normal limb.

DISCUSSION

The sensation of vibration is the most delicate known test of the integrity of the posterior columns of the cord. The great advantage of testing according to Symns' technique, which is rarely carried out, is that a quantitative result is obtained, thereby providing an indication of small changes, and giving a graph, which can be compared with the results of testing the patient at some future date.

The sensation of vibration is definitely related to that of deep pressure and passive movement, and the sensation, especially at the sacrum, shows a correlation with the presence or absence of Rombergism. To a less extent the sensation can be felt in the skin, like the sensation of touch, and this small component of the sensation can be abolished by local anæsthesia.

The fork is conveniently applied to some bony prominence, as thereby the bone is made to vibrate and transmit the impulse through the soft tissues. Disease or fracture of bones has little effect on the duration of the sensation, and probably only a small part of the sensation, if any, is felt in the bony tissues themselves. The sensation is perceived in the cerebrum—a protopathic element in the thalamus, and an epicritic in the cortex.

With practice, in a normal individual, the acuity of the perception can be considerably increased, and is found to be so increased in healthy blind individuals. The sensation is diminished by fatigue, of which there are two forms—extrinsic fatigue, due to tiring of the organism as a whole, and intrinsic fatigue, due to overstimulation of the sensation of vibration itself. It is the neglect of this latter factor that is the chief error in testing by Symns' method, and a simple way of overcoming this has been devised.

It has been frequently stated in the literature that diminution of the sensation at the sacrum denotes a lesion of the cord, whereas diminution at the limbs and a normal sensation at the sacrum mean a peripheral neuritis. With the latter assertion I have no dispute, but it is impossible to accept the view that absence of the sensation at the sacrum necessarily means a cord lesion. Were it so five of my cases of diabetes would have pure cord lesions, and only two pure peripheral neuritis; three out of five cases of microcytic anæmia would have cord involvement, whereas only one had suggestive signs and symptoms; and a case of syphilitic aortic disease, with no signs of symptoms of nervous lesions, and a perfectly normal cerebrospinal fluid, would also be diagnosed as such. Nevertheless, absence of sensation of vibration at the sacrum is suggestive, and should lead to further investigation. It should not be forgotten that the presence of a large sacral pad of fat is an element in reducing sacral acuity.

Probably the greatest help which the tuningfork gives is in the diagnosis of pure motor lesions, such as acute anterior poliomyelitis, amyotrophic lateral sclerosis, progressive muscular atrophy, and their differentiation from combined lesions, such as multiple neuritis, transverse myelitis, disseminated sclerosis and subacute combined degeneration of the cord. But note must be taken of the diminution of the acuity with age.

SUMMARY

1. A brief résumé of the history of the study of the sensation is given.

2. A few physiological principles involved in the perception of the sensation and its course in the brain and cord are discussed.

3. Ten individuals without disease of the nervous system were examined to correlate the normal with the investigations of others.

4. Five people above the age of 70 were examined to demonstrate the loss of vibratory acuity with age.

5. Five blind but otherwise healthy individuals were examined, and their vibratory acuity found to be above the normal, especially in the limbs.

6. Five cases of syphilitic aortitis were examined; sensation at the sacrum was absent in two, and the central nervous system diseased in one of these.

7. Five sufferers from postencephalitic Parkinsonism were tested, and their curves found to be rather low, but well within the normal. The feature of the curves was their evenness.

8. Five tabetics were examined, and their curves found to agree with the typical curve as described by Symns, showing the 'sacral dip.' One of the individuals had the lowest sensibility yet tested.

9. In four out of five cases of disseminated sclerosis there was marked diminution of acuity. The sensation of vibration is often the only sensation disturbed in this disease.

10. Five cases of microcytic anæmia are discussed.

11. Of 25 cases of diabetes 48 per cent. showed lesions of the nervous system. Fourteen had deficient sensation of vibration at the sacrum, but this could not have meant a cord lesion. In about half the cases of diminution of the vibratory sensation the loss would never have been found had not Symns' technique been used.

12. A brief résumé of the history of nerve lesions in pernicious anæmia is given. In subacute combined degeneration of the cord the sensation of vibration is deranged more frequently than any other sensation. Of the 25 cases tested 72 per cent. showed nervous lesions. In two-fifths of the cases the loss was quantitative and would not have been disclosed had not a quantitative technique been used. Four cases, or 16 per cent., had peripheral neuritis without cord involvement.

13. A few miscellaneous cases to illustrate various points are presented. One of these, a case of intracranial neoplasm, had the highest vibratory acuity so far encountered.

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BILATERAL ATROPHIC LOBAR SCLEROSIS FOLLOWING THROMBOSIS OF THE SUPERIOR LONGITUDINAL SINUS

By

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INTRODUCTION

In the category of atrophic lobar sclerosis it is customary to group a number of degenerative affections of the cerebral cortex having a differing and often obscure pathogenesis but a comparatively uniform morphology. Macroscopically, the cortex in these cases shows a shrinkage of the gyri with a corresponding widening of the intervening sulci. Apart from the changes produced by these distorting factors, the general convolutional patterning of the hemispheres is normal, so that usually it is possible to distinguish by naked-eye inspection this type of abnormality from true microgyria of developmental origin. The name 'ulegyria' is often used to describe this condition of atrophy of the convolutions. Microscopically, it is apparent that the neurones of the affected gyri have degenerated to a greater or less degree and that an extravagant proliferation of the fibrous neuroglia is present in those areas where neuronal destruction has been most complete. This gliosis tends to vary according to the time of onset of the primary cortical lesion, being densest and most widespread when the disease has started during fœtal or infantile life and being less pronounced in the more mature brains.[15] In the latter class of case there is evidence both from the clinical and pathological standpoints that a progressive degeneration of the cortex may occur. In this type of progressive cortical atrophy the lesions are essentially laminar in distribution and affect chiefly the third or pyramidal cell-layer.

As Hallevorden [8] has pointed out, the similarity of the histopathological findings in cases of lobar sclerosis is no proof of an uniform ætiology. This observation is particularly pertinent when an attempt is being made to evaluate the part played by birth injury in the ætiology of a given case. Although the extensive investigations of Schwartz [13] have shown that atrophic sclerosis may follow multiple small intracerebral hæmorrhages or softenings, it is clearly unjustifiable to classify all cases of lobar sclerosis with a clinical history of 'difficult labour' as the result of vascular lesions sustained during birth. Moreover, the precise mechanism by which these hæmorrhages are produced is not yet fully understood. According to Schwartz, they

result from an intense venous engorgement and stasis of blood in the presenting fœtal head, this congestion being brought about by the difference between the atmospheric pressure acting upon the presenting head after rupture of the membranes and the higher intrauterine pressure acting upon the body of the fœtus. This ' suction ' theory has been severely criticized by Rydberg.[11] In this author's opinion the areas of softening and bleedings commonly found in the brains of stillborn infants are the result of a failure of the arterial blood pressure to compensate for the excessive rise in the general intracranial pressure due to prolonged or sudden compression of the fœtal head.

In spite of this conflict of opinion, a valuable outcome of these modern investigations has been to emphasize the importance of an intact vascular supply to the developing cerebrum. This fact is exemplified in the following case of atrophic sclerosis of the cortex associated with obliteration of the superior longitudinal sinus and apparently occurring at the time of birth. Thrombosis of cortical veins, later spreading to the superior longitudinal sinus, was considered by Gowers [7] to be a common cause of infantile cerebral palsy, but there are few detailed pathological observations to substantiate this view. The case about to be described seems, therefore, to be worthy of record as an example of this type of vascular lesion. Finally, it may be said concerning birth injuries in general and this case in particular that although the clinical history and pathological findings may strongly support the view that the cerebral damage has been produced by the vascular disturbances of parturition, it is an open question whether or not prenatal factors have not been coincidently operative. Individual variation in angioarchitectonics may well play an important but as yet undemonstrable part in the production of these vascular lesions of early life.

CLINICAL ASPECT

Family History.—The patient was the male first born of a family of three, the other two sibs being reported as normal. All three sibs were post-mature. Father and mother were physically and mentally normal and in comfortable financial circumstances. There was no history of nervous disease in the parents or grandparents.

The Pregnancy.—This was of 10 months' duration, and according to the mother labour pains and a partial dilatation of the os uteri occurred exactly four weeks before the child was born. Four months before the birth the mother suffered from an intractable dermatitis, ' like masses of boils,' affecting the thighs and abdomen. A bacteriological examination was made and streptococci only were found. Some months after the birth it was found that the mother was suffering from bad pyorrhœa and an apical abscess of a tooth.

Birth History.—The patient was delivered by head presentation after a dry labour lasting seven hours. Forceps were not used. The child was badly asphyxiated from aspirated meconium and gave hardly any sign of life, but was revived by oxygen and hot and cold baths. No cry was uttered for three hours.

Postnatal History.—There was no rigidity present in the limbs immediately after birth, but no spontaneous movements were made. Twice on the second day the infant went black in the face and breathing was difficult; after this he slept for 15 hours. He was unable to take the breast. By the fourth week signs of stiffness were

present in the hands ; later this was observed in the arms and then in the feet. Subsequently, in spite of massage and training, no appreciable improvement in the spasticity occurred. Increasing spasticity, especially in the lower limbs, was observed up to about the age of three. No remission or sudden exacerbation of the general condition occurred, and he suffered from no febrile illness during infancy.

Convulsions started when he was about six weeks old, slight limb contractions being associated with a convergent squint and a jerking forward of the head. In the early days these fits were frequent—every two or three hours—but seldom at night. They occurred on waking after a sleep during the day time. The last fit occurred at the age of nine months.

Dr. H. Cohn of Edinburgh saw the child when he was four years old, and has kindly supplied the following information :

He was poorly nourished, length and weight corresponding to a boy of three years of age. Unable to stand or to speak. The head sank forwards when not supported and when supported there was a marked tendency to turn the head to the side, more often to the right. There was marked salivation and dribbling.

Cranial Nerves.—Except for a slight paresis of the right abducent nerve, eye movements were normal. He could follow a moving finger. The corneal reflexes were present. There was slight paresis of the right side of the face and sometimes involuntary movements of the face produced an expression similar to risus sardonicus. The glossopharyngeal-vagal reflex was diminished and imperfect swallowing sometimes occurred. The tongue showed no paresis, but occasional involuntary movements were observed.

Motor System.—There was marked but variable spasticity of both upper limbs, the tendon-jerks being much increased. Erb's and Hoffman's reflexes of the fingers were present, as was also Tromner's phenomenon. The abdominal and cremasteric reflexes were present on both sides. There was a severe degree of spasticity in the lower limbs, especially in the adductor muscles, so that the right foot usually crossed the left. Knee- and Achilles-jerks were very much increased. No true clonus was obtained. Phenomena of Babinski, Rossolimo, Bechterev and Oppenheim were present on both sides.

Sensation.—Response to pinpricks over the body surface was obtained.

On admission to Stoke Park Colony on December 21, 1934, the patient was aged five years and seven months. His height was 3 feet and he weighed two stone. There was a general brownish pigmentation of the skin and pallor of the mucous membranes. He was unable to stand or sit unaided. He lay in an attitude of general flexion with the legs crossed above the knees. There was a marked rigidity of the upper limbs which to some extent masked the tendon reflexes. The lower limbs also were very rigid and the kneejerks and anklejerks increased, the former being obtained by striking the subcutaneous surfaces of the tibiæ all the way down the bone. The plantar reflexes were flexor (Babinski's sign negative). The abdominal reflexes were not obtained. There was incontinence of fæces and urine. Since he was speechless, helpless, and unable to make any attempt at simple performance tests he was classified as an idiot.

Terminal Illness.—Five days after admission the temperature rose to 100·2°. There was a swelling of the lower lip with an abrasion of the mucous membrane as though the patient had bitten himself. On the evening of the next day the patient suddenly collapsed, the colour being very poor and the breathing distressed. He vomited a little dark brown fluid. On examination he was found to be in a comatose condition. There was bilateral divergent strabismus, pinpoint pupils, and absent tendon-reflexes. Corneal reflexes were present. There was no rigidity of neck muscles. The heart rate was rapid (120) and the sounds poor. The urine contained a trace of albumen. Later the temperature rose to 103·4° and he died the same evening.

COMMENT

So far as the history of this case throws light upon probable causative factors, it would seem that the most significant fact is the attack of respiratory distress and subsequent somnolence that occurred on the second day after birth. It is this piece of information, rather than the history of dry labour and asphyxiation, that is suggestive of some cerebral vascular lesion resulting in bleeding or softening. Rydberg has emphasized the importance of this type of history in cases of fatal birth injury. He made a careful examination of the brains of 75 infants who died within 10 days of birth. He found 34 in which considerable intracranial bleedings or softenings had occurred. All these fatal cases showed a clinical picture which is 'perhaps not pathognomonic of intracranial bleedings and primary injuries to the brain substance but is very nearly so. It is characteristic of such cases that there is a symptom-free interval in the first hours or days after birth, that the children fall ill rather suddenly and that then the symptoms of cerebral irritation play an important part during the acute stage of illness. In this group are placed (1) cases which show cerebral motor symptoms such as twitchings, spasms and pronounced general rigidity, and (2) cases which do not present any motor symptoms if the child exhibits a disturbance in the breathing, cyanosis, or a pronounced drowsiness, and a clearly increased tension of the large fontanelle is observed at the same time.'

While such intracerebral bleedings and softenings have evidently a highly lethal effect, some of the individuals in whom they occur must survive, and it is a matter of importance to distinguish this class of case from patients whose spastic paralysis is the outcome of cerebral agenesia. A mere history of difficult labour is not, of course, a sufficient reason for attributing the cause of the diplegia or mental defect to birth injury. Several investigations, notably that of Dayton,[5] have shown that the prognosis for children born after abnormal labour differs little from that for normal birth. A history of difficult labour is, in fact, common; on the other hand, infantile cerebral palsies are rare. The important point in evaluating a history of birth injury is not the mere fact of abnormal parturition but the history of the characteristic signs of intracranial injury.

There is, therefore, in the present case clinical evidence which suggests that some vascular lesion occurred at or shortly after birth.

MORBID ANATOMY

Post-mortem Examination.—Immediately after death 10 per cent. formol-saline solution was injected into the skull through the orbits and the cisterna magna, as much cerebrospinal fluid as possible having been previously withdrawn. An autopsy was made about 12 hours after death. The only abnormalities found were the following: The lungs showed congestion of the posterior part of the lower lobes. The skullcap was thin and poorly ossified.

The dura mater was immovably adherent to the parietal bones and in this situation the lumen of the superior longitudinal sinus was obliterated by dense adhesions. No signs of old hæmorrhages were present. The pia arachnoid membranes over the vertex of the brain were thickened and pinkish in appearance, but not adherent to the cortex. The vessels were very engorged.

The brain and spinal cord were removed and suspended in 10 per cent. formol-saline, representative blocks having been taken for fixation in formol-ammonium bromide solution.

Macroscopic examination of the brain after stripping the meninges showed that a condition of atrophic sclerosis was present in the gyri of the convexity adjoining the left paracentral lobule. The upper half of the left precentral, the upper third of the postcentral gyrus, and the part of the superior parietal lobule adjacent to the postcentral sulcus were markedly shrunken. The superficial puckering of these gyri gave the appearance of verrucous atrophy. This sclerosed region was not uniformly hard to the touch, for the superficial tissue was soft and friable compared with the increased density of the subjacent parts. This peculiarity was especially noticeable when fresh blocks were being cut out for formol-bromide fixation, and it was found that this deepseated toughness extended in the white matter over a much larger area than might have been supposed from the surface extent of the shrunken gyri. The right hemisphere was macroscopically less affected. Here the area most involved was the postcentral gyrus, the upper half of which was very narrow and of increased density. Posteriorly, this sclerosed gyrus was overhung and partially concealed by the adjoining superior parietal lobule. Apart from the distortion introduced by this apparent shrinkage and sclerosis the general convolutional patterning of the hemispheres exhibited no gross abnormality in the fissuring.

Microscopical Examination.—The following abnormalities were found in the representative areas of the central nervous system that have been examined (for an account of the areas selected and the methods used see Appendix).

1. *The Meninges.*—(*a*) The dura mater surrounding the patent part of the superior longitudinal sinus was normal. (*b*) The leptomeninges of the vertex of the brain showed simple fibroblastic thickening. The pial vessels were very engorged and some recent capillary hæmorrhages were seen.

2. *The Cerebral Cortex.*—(*a*) Extensive areas devoid of neurones were found in many of the cortical areas selected for examination. The macroscopically shrunken gyri showed this abnormality most obviously in the neighbourhood of the great longitudinal fissure. In this situation the whole grey matter cortex showed a severe diminution in the number of neurones, and both paracentral gyri exhibited areas of clearing in the third and fifth layers (fig. 1, A).

In the upper and middle portions of the precentral gyrus the neuronal defects were chiefly localized in the third or pyramidal cell-layer. The post-

central gyrus and superior parietal lobule of the left hemisphere suffered in a similar way except that the granular layer (L iv) was equally affected. All

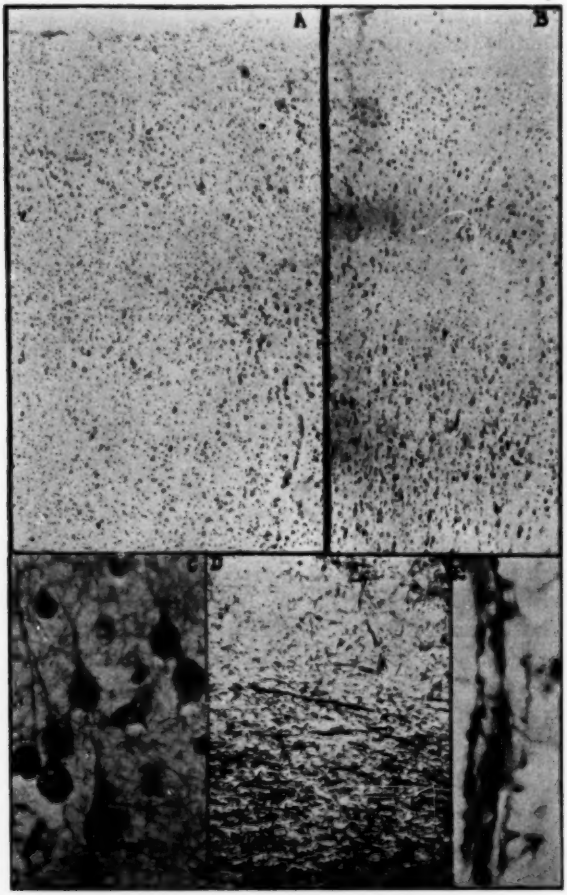

Fig. 1. A. Left paracentral lobule. Areas of cortical obliteration. Toluidin blue × 38.
B. Left upper postcentral gyrus. Laminar defect in L iii. Toluidin blue × 30.
C. Primitive nerve-cells in wall of sclerotic precentral gyrus. Bielschowsky's stain × 475.
D. Wall of postcentral gyrus. Anderson's modified Kulschitsky-Pal stain × 48.
E. Wavy and beaded myelinated fibres in paracentral lobule. Stain as in D × 475.

these cortical areas gave the appearance of being incompletely divided into two parts by an a-neuronal strip in the middle of the grey matter containing only an excess of glial nuclei (fig. 1, B).

Smaller defects in the pyramidal cell-layer were found in other parts of the frontal lobe (FB, FC, FD), in the temporal lobe (TA, TG), and to a less

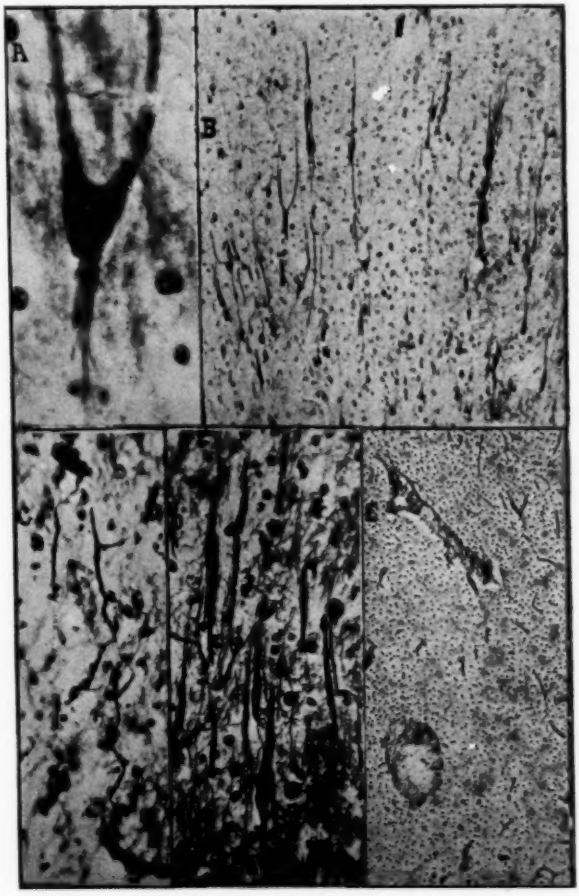

Fig. 2. A. Abnormal type of neurone from paracentral gyrus. Hortega's fourth variant of the tannic silver method × 475.
B. Abnormal neurones in precentral gyrus. Cajal's gold sublimate method × 63.
C. Mesenchymal reticulin fibrils in a sclerotic area of the cortex. Perdrau's method × 214.
D. Swollen and agglutinated axiscylinders in paracentral gyrus. Bielschowsky's stain × 214.
E. Fibrotic precapillary and mesenchymal proliferation in sclerotic white matter. Hortega's silver carbonate method for astrocytes × 63.

degree in the visuopsychic area (OA). The frontal pole (FE) and the visuosensory area (OC) were intact.

These neuronal defects were most frequently seen in the walls of the gyri, the crowns being unaffected save in the shrunken parts of the cortex. The nerve-cells in the deeper parts of the walls of the gyri were often represented as mere clusters or columns of small cells little better than neuroblasts. The Sommer sector of cornu ammonis showed a marked diminution in the cells of the pyramidal layer.

(*b*) Primitive and abnormal neurones were present in the above-mentioned sclerotic parts of the cortex. Isolated clumps of nerve-cells which were developed little better than neuroblasts occurred chiefly in the deeper parts of the walls of the gyri (fig. 1, C). In addition to these primitive cells, peculiarly elongated or Y-shaped forms were found in the proximity of the areas of cortical clearing (figs. 2, A, and 2, B). Such cells were not seen elsewhere in the cortex. In the sclerotic grey matter the axiscylinders occasionally exhibited thickening and agglutination reminiscent of senile changes (fig. 2, D).

(*c*) *Glial changes.*—There was marked sclerosis of the paracentral lobule, the upper pre- and postcentral gyri and adjoining superior parietal lobule. The overgrowth of fibrous neuroglia was patchily distributed between the remaining groups of nerve-cells and seemed to be denser around the blood-vessels (fig. 3, D).

In the grey matter a laminar gliosis corresponding to the laminar neuronal outfall was often a conspicuous feature (fig. 3, A). The subpial felt work was thickened and strands of glial fibres were often seen passing between the surface of the gyrus and the deeper cortical layers. Here and there small cystic areas containing phagocytic cells were present in the more sclerosed gyri.

The white matter of the affected and neighbouring gyri and also the subjacent centrum ovale exhibited a pronounced gliosis of isomorphous type. Silver methods showed the number of astrocytes per field to be increased in the white matter of the less affected gyri (fig. 3, C). Examples of large fibre-forming macroglia were frequently demonstrated (fig. 3, B).

The oligodendroglia exhibited acute swelling with little intracytoplasmic content.

The microglia was present in normal forms in the unaffected parts of the cortex. In the small cystic areas mentioned above, compound granular corpuscles were present. An occasional elongated bipolar rod-cell was found in the frontal cortex adjoining the sclerotic areas, but there was no microglial mobilization.

(*d*) *Changes in the myelin.*—The radial and supraradial fibres in the sclerotic parts of the cortex were very poorly and patchily represented. The myelin sheaths exhibited numerous varicosities and their course was somewhat wavy (fig. 1, E). In the postcentral gyrus a similar reduction in fibres had occurred, though even in this distorted area a few of the normal horizontal fibres of large calibre were demonstrable (fig. 1, D). No ' plaques fibro-myéliniques ' were seen.

(*e*) *Changes in the blood-vessels.*—In the neighbourhood of the cortical areas characterized by the absence of nerve-cells the capillaries often showed

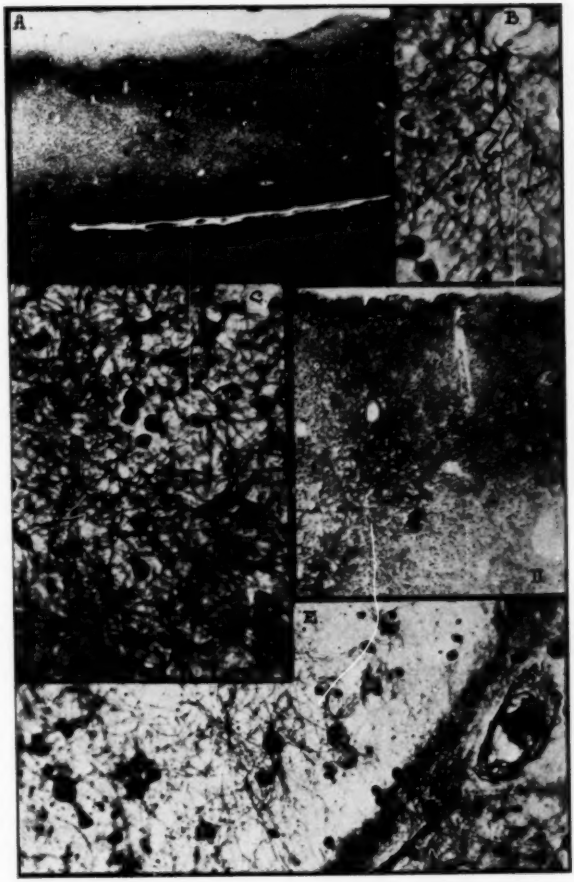

Fig. 3. A. Laminar gliosis in wall of postcentral gyrus. Holzer stain × 15.
B. Type of large fibre-forming astrocyte found in less affected cortex (area FC). Hortega's method for astrocytes × 475.
C. Gliosis in white matter of frontal cortex. Area and stain as in B × 214.
D. Gliosis in paracentral gyrus. Holzer stain × 63.
E. Paracentral gyrus. Phagocytic cells in cystic areas. Vessel at the right shows proliferation of mesenchymal fibrils. Stain as in B × 290.

considerable fibrosis. Here also occurred an increase in visible vessels which was well demonstrated in sections stained by silver methods. In the areas of severe gliosis an overgrowth of the mesenchymal reticulin fibrils of the

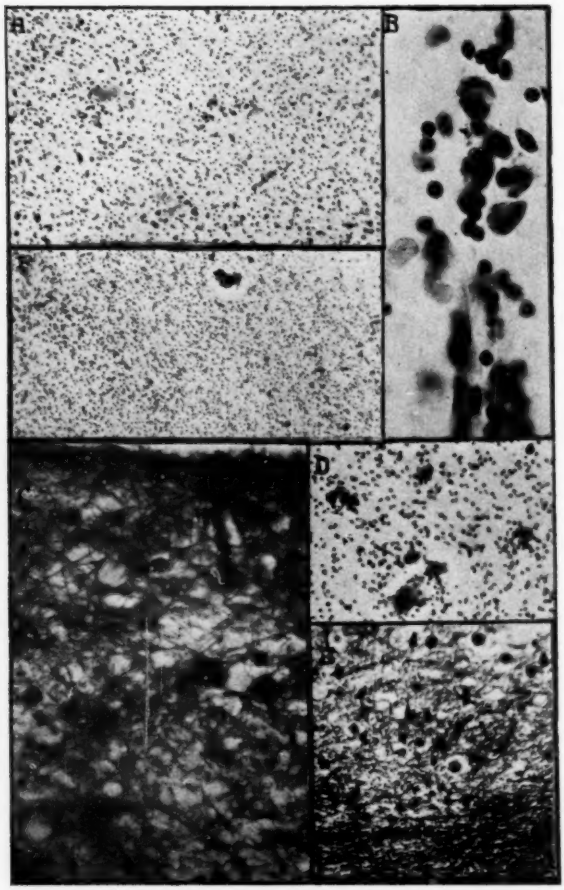

Fig. 4. A. Left putamen. Outfall of neurones and increase in glial nuclei. Toluidin blue × 48.
B. Phagocytic cells in putamen. Toluidin blue × 475.
C. Gliosis in putamen. Holzer stain × 475.
D. Thalamus. Calcified neurones in lateral nucleus. Glial increase. Vessel with phagocytic cells. Toluidin blue × 63.
E. Wall of precentral gyrus. Deposits of lipoid nature. Anderson's modified Kulschitsky-Pal stain × 79.
F. Thalamus. Dense glial proliferation and gross reduction in neurones. Toluidin blue × 48.

blood-vessels had occurred (figs. 2, C, and 2, E). Recent small capillary hæmorrhages were occasionally observed.

3. *The Basal Ganglia.*—(*a*) The thalamus showed an intense gliosis and a gross reduction in the number of neurones in the medial and lateral parts

of the organ (fig. 4, F), the pulvinar and anterior nucleus being relatively well preserved. The nerve-cells remaining in these sclerosed areas were often

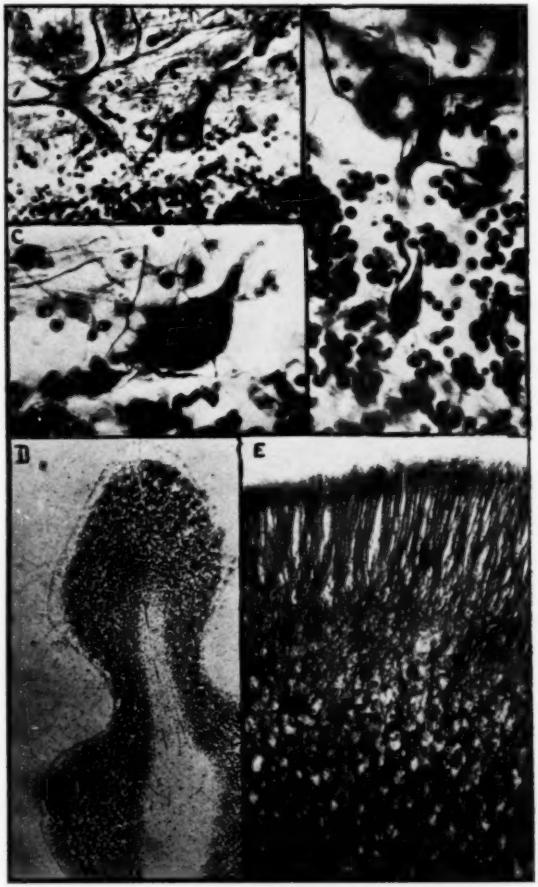

Fig. 5. A. Superior vermis of cerebellum. Hypertrophic antlering of the dendrites of Purkinje cell. Bielschowsky's stain × 214.
B. As in A. Neuritic torpedo. Thickened basket network. × 475.
C. As in B. × 475.
D. As in A. Paucity of Purkinje cells. Increase in nuclei of Bergmann's glia. Toluidin blue × 29.
E. Gliosis in superior vermis. Holzer stain × 214.

calcified (fig. 4, D). The myelinated fibres in these affected areas were densely packed together. No status marmoratus was seen.

(*b*) The putamen also showed a diminution of nerve-cells and an over-

growth of fibrous neuroglia (figs. 4, A, and 4, C). The vessel walls contained much fat and compound granular corpuscles were present in their vicinity

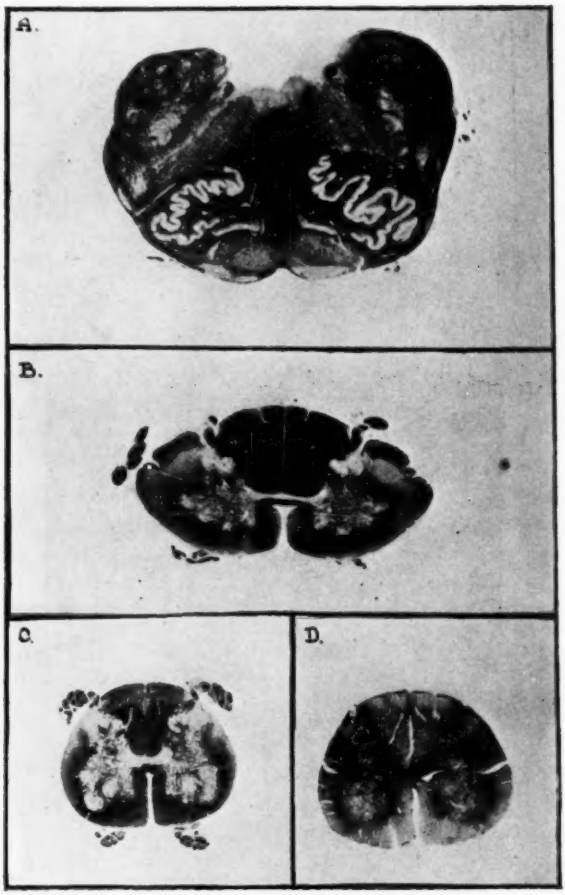

Fig. 6. A. Medulla oblongata. Degeneration of pyramids. Anderson's modified Kulschitsky-Pal stain × 3.
B. Cervical enlargement. Degeneration of lateral columns. Stain and magnification as in A.
C. Lumbar enlargement. As in B. × 3.
D. Lumbar enlargement. Gliosis in lateral columns. Holzer stain × 3.

(fig. 4, B). There were a few microscopic areas devoid of myelinated fibres and occasional coarse varicose fibres were present.

(*c*) The globus pallidus was normal apart from a slight crowding together

of the nerve-cells. No abnormality in neurones, glia or myelin was found in either the head or body of the caudate nucleus.

4. *The Cerebellum.*—In sections prepared from the superior vermis it was obvious that some folia were very deficient in Purkinje cells (fig. 5, D). In these areas, which were the exception rather than the rule, the nuclei of the Bergmann glia-cells formed a distinct layer leaving a clear zone devoid of Purkinje cells in the subjacent parts of the folia. The radial glial fibres of the molecular layer were thickened and increased in number (fig. 5, E). Abnormalities were present in some of the Purkinje cells. These took the form of hypertrophy of the dendritic antlers, hypertrophy of the basket networks and the presence of neuritic torpedoes (figs. 5, A, 5, B, and 5, C). The lateral lobes also showed a diminished number of Purkinje cells and glial overgrowth in some folia. In these parts the changes were less prominent than in the superior vermis. The central white matter of the cerebellum showed an excess of neuroglia-fibres and stained a darker shade with the Holzer method than did controls. Round the cells of the dentate nucleus there was a notable increase in glial nuclei. Myelinization of the central white matter appeared to be adequate.

5. *The Condition of the Pyramidal Tract.*—In the cerebral cortex the Betz cells appeared to be reduced in numbers owing to the defects present in the deeper layers of the paracentral lobules and upper precentral gyrus. Nevertheless, staining for myelin showed no obvious abnormality in the left internal capsule, crus or pons. At the level of the medullary olives the pyramids were smaller than normal and showed a definite thinning of myelinated fibres and an excess of glia (fig. 6, A). This defect in myelinization and coincident glial hyperplasia could be traced in the lateral columns down to the lumbar enlargement of the spinal cord (figs. 6, B, 6, C, and 6, D).

6. *Products of Degeneration.*—(*a*) Fat was present in excess in the vessel walls of the thalamus and putamen where phagocytic cells were present. The calcified nerve-cells of the thalamus showed numerous fatty globules adsorbed to their surfaces. The fatty granules present in the nerve-cells of the cerebral cortex appeared to be within normal limits.

(*b*) In the sclerotic parts of the cerebral cortex there were numerous small crumb-like deposits staining greyish-black with the Kulschitsky-Pal stain and resisting differentiation. They were confined to the deeper layers of the grey matter. They were not demonstrable by van Giesen staining and are thus not examples of ' colloid ' degeneration (fig. 4, E).

(*c*) A large number of metachromatic granules were present in the internal capsule.

7. *Capillary Hæmorrhages.*—These were frequently seen in many parts of the nervous system. These bleedings were microscopic in size and consisted of unchanged red blood corpuscles. Slightly larger bleedings were present in the substantia reticulosa of the pons in the midline.

DISCUSSION

Although the cortical changes described above are in the main typical of atrophic lobar sclerosis,[12] the nerve-cell picture presented by those gyri showing extensive defects in the third layer might, perhaps, be confused with the four-layered type of cortex described by Bielschowsky in certain cases of microgyria.[1] This latter condition, however, is a pure malformation brought about by the defective migration of neuroblasts from the matrix to the cortex. The neurones eventually become distributed in two main laminæ, a cell-free layer of myelinated fibres intervening between them. The superficial layer of these nerve-cells is usually arranged in a wavy or garland-like way (internal microgyria). The absence of fibrous gliosis in the grey matter of the cortex, the characteristic myelin picture and the grossly abnormal convolutional pattern of the brain make this type of microgyria readily distinguishable from the cortex in lobar sclerosis, where the abnormalities have been produced by a process of destruction, not of abnormal development. That such a destructive process has been operative in the present case is indicated by the dense gliosis in areas of the cortex devoid of neurones, the disturbance in the myelin picture, the presence of small groups of scavenger cells, and the proliferation of the mesenchymal fibrils of the blood-vessels. The arrested or perverted development of some of the remaining neurones in the affected areas is to be explained as the reaction on the part of immature cells to a grossly altered environment. In a like manner the extensive gliosis in the white matter may be attributed to the result of the stimulus to proliferation provided by disintegration products derived from foci of neuronal degeneration. Such findings are commonly found as sequelæ to lesions sustained by the immature nervous system during the period of anatomical plasticity.

The autopsy findings showed in the present case that the bilateral atrophic sclerosis of the cortex was associated with partial obliteration of the superior longitudinal sinus. According to Collier and Adie [4] 'there is no other way out for the blood of the convexity of the hemispheres than by the superior longitudinal sinus, and it is for this reason that blocking of this sinus always results in disastrous bilateral softening of the hemispheres.' There is good reason for believing, therefore, that in this patient the cortical lesions have followed the sinus thrombosis, and, furthermore, since the child suffered from no sudden exacerbation of the general condition or from any illness in infancy, that the thrombosis did not occur later in life than the second day. The histological examination of the affected gyri showed that the severity of the destructive process diminished in a regular manner as the cortical areas adjoining the Sylvian fissure were approached until, finally, only small areas of neuronal obliteration were found in the lower parts of the walls of the convolutions. In other words, the parts of the brain most affected by the congestion and anoxæmia following the sinus thrombosis were those in

which there was the least chance of the blood being led off by other channels than the superior longitudinal sinus—for example, by the inferior cerebral veins. The pathological process by which the neuronal destruction had been brought about was therefore somewhat similar to that claimed by Schwartz to be operative in some cases of birth injury to the brain, namely, vascular stasis following intense venous engorgement. It is significant that in the present case the layers of the cortex most affected (L iii and L iv) were those in which the blood supply is normally the most abundant. It would seem that these neurones require the most abundant oxygen and that they suffer most by its deprivation.

The structural abnormalities found in parts of the nervous system remote from the cerebral cortex may be adequately accounted for as degenerative changes secondary to the primary cortical lesion. Biemond [3] has demonstrated that atrophy of the cerebellar vermis may result from a lesion in the pons affecting the corticopontocerebellar fibres. The intimate connexion existing between the neopallium and cerebellum gives one good grounds for believing that the patchy atrophic change found in the cerebellum of the present case was secondary to the primary cortical destruction. Similarly, the remarkable poverty of neurones and the intense gliosis found in the medial and lateral parts of the thalamus are to be interpreted as secondary manifestations. A strictly comparable condition was found by Bielschowsky [2] in his cases of cerebral hemiatrophy. Here the essential cortical lesion consisted in a widespread atrophy of neurones in the third layer of the cortex. He interpreted the areas of thalamic atrophy found in these brains as the result of a retrograde degeneration of the thalamocortical neurones consequent upon the destruction of the receptor layers of the cerebral cortex. The extensive involvement of the postcentral gyrus in the present case is strong evidence that a similar process was operative in producing a secondary atrophy of the thalamus. The comparative richness of the myelinated fibres in the atrophic subcortical areas is to be accounted for by the crowding together of the remaining axones owing to the shrinkage of the tissues.

The condition of the pyramidal tracts in this case deserves special mention, since the histological findings enable one to date roughly the onset of the cortical degeneration. It is necessary to reconcile two apparently contradictory facts revealed by microscopical examination. In the first place, no defect in myelinization of the pyramidal tracts was demonstrated in the pons, midbrain, or internal capsule. This observation does not support the view that a simple descending degeneration of the pyramidal fibres had followed destruction of the cells of origin in the infragranular cortex unless, indeed, the outfall of fibres was too limited to be clearly shown by the methods employed. Secondly, in the medulla and spinal cord there was present the typical picture of a demyelinization of the pyramidal tracts, namely, a diffuse outfall in myelinated fibres and an accompanying fibrous

gliosis. The latter finding is evidence in favour of degeneration, not simply arrest of myelinization. An explanation of these apparently contradictory observations may be found when the distribution of the cortical lesions in this case is compared with that found in the variety of lobar sclerosis producing the remarkable condition of 'intracortical hemiplegia with intact pyramidal tracts.' In this type of cortical degeneration, several examples of which have been recorded in children and adults,[2 6 9 10 12 14] a progressive atrophy of the pyramidal cells of the third cortical layer takes place, the infragranular cortex and the Betz cell-layer of the motor cortex remaining relatively intact. The result of this selective atrophy is that transcortical receptor impulses are prevented from reaching the effector projection-system by which voluntary movements are normally exteriorized. A clinical condition similar to ordinary hemiplegia is thus set up as this laminar degeneration of the cortex progresses, although no degeneration of the pyramidal tract is found owing to the fact that the cells of origin in the infragranular cortex are preserved. Now in the present case a similar laminar defect of the third layer of the cortex was present, but the peripheral portion of the pyramidal tracts exhibited degenerative changes. May it not be that this difference is due to the respective ages at which the atrophic cortical changes began? In the children and adults in whom intracortical hemiplegia developed the pyramidal tracts had attained a degree of maturity which was not present in the case now being described. Normally, the pyramidal tract is incompletely myelinated at birth. This patient was a post-mature infant, so that it is reasonable to assume that myelinization had advanced somewhat more at the time of birth than would normally be the case. Nevertheless, the caudal part of the tracts would be still very imperfectly myelinated. Although an adult pyramidal tract is apparently able to withstand the abnormal situation resulting from the cessation of corticofugal impulses, it does not follow that incompletely myelinated fibres are equally robust. On these grounds, therefore, it seems probable that the degeneration of the pyramidal tracts in the present case was not a simple descending demyelinization, but rather an absorption of incompletely myelinized fibres resulting from a cessation of corticofugal impulses.

It follows from the above discussion that the time of onset of the thrombosis occurred not earlier than the end of fœtal life—i.e. at a time when the pyramidal tract axones had penetrated the spinal cord but were still imperfectly myelinated. The birth history, as has been pointed out, suggested that an intracranial lesion had been sustained at birth and that an extension of the damage had occurred on the second day. Nevertheless, it is possible that this unusual accident may have been facilitated by prenatal factors. Apart from a developmental anomaly of the sinus itself, the possibility cannot be ruled out that the streptococcal infection of the mother during pregnancy may have acted as a predisposing influence in producing the thrombosis. The passage of cocci into the fœtal circulation, their lodgement in the wall of

the superior longitudinal sinus, and the ensuing toxic action upon the endothelium might undoubtedly form a focus for future thrombus formation.

Finally, it is readily understandable how the pathological changes in this brain have resulted in the clinical condition of idiocy. In the first place, the defect in the effector apparatus prevented the normal exploratory reactions of the infant; secondly, the lesions of the postcentral gyri shut out the kinæsthetic impulses the synthesis of which with exteroceptive sensory elements in the parietal association areas is the essential basis of cognition and behaviour. This latter consideration accounts for the lack of improvement to remedial training shown by the patient.

My thanks are due to Professor R. J. A. Berry, the Director of Medical Services at Stoke Park Colony, for permission to publish this case and for much helpful advice; to Dr. A. L. Taylor, Pathologist to Bristol General Hospital and to the Colony, with whom I have discussed many aspects of the work; and to Dr. Cohn, who kindly supplied notes as to the neurological findings prior to the patient's admission to the Institution.

APPENDIX

Areas Examined and Methods Employed

The Cerebral Cortex.—With the exception of the right paracentral area, the left cerebral hemisphere only was examined. The following representative areas were studied: the upper middle and lower parts of the pre- and postcentral gyri; the paracentral gyrus; the frontal association areas (FB, FC, FD, FE of von Economo); the superior parietal lobule; the angular and supramarginal gyri; the visuopsychic and visuosensory areas; the temporal pole and superior temporal gyri; and the hippocampal convolution. Larger horizontal survey sections of the frontal and occipital lobes were also examined.

From *the diencephalon* of the left side sections have been prepared from the thalamus, putamen, globus pallidus, and head and body of the caudate nucleus, and also larger celloidin sections displaying the internal capsule in this situation.

The midbrain at the level of the emergence of the third cranial nerve, the *medulla* at the level of the middle of the olives and of the decussation of the pyramids, the *upper cervical* region and *cervical enlargement, midthoracic and lumbar enlargement of the spinal cord* have also been examined.

In the cerebellum sections have been studied from the superior vermis and lateral lobes.

The following staining methods have been adopted:

1. For Nissl's granules	toluidin blue.
2. For neurofibrils	Bielschowsky's stain.
3. For macroglia	Warkany-Holzer method, Cajal's gold sublimate and Hortega's silver carbonate method for astrocytes.
4. For oligodendroglia and microglia	Hortega's method and the modification of Cone and Penfield.
5. For myelin	Anderson's modification of the Kulschitsky-Pal method, and osmic acid.
6. For fatty substances	Scharlach R. and Marchi's method.

Occasional use has also been made of Perdrau's modification of the Bielschowsky method and of Hortega's fourth variant of Achucarro's tannic silver process.

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DEGREES OF AUTOMATIC ACTION: SOME PSYCHIATRIC APPLICATIONS OF HUGHLINGS JACKSON'S CONCEPT OF 'REDUCTION TO A MORE AUTOMATIC CONDITION'

By

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INTRODUCTION

The doctrine of the reflex constitution of the nervous system—the foundation of Hughlings Jackson's neurological teaching (II, 63, 399)*—has received ample recognition in the study of the more primitive activities of the organism, such as locomotion, but has, by contrast, received practically no recognition in the study of that most complex activity, mentation. Failure to regard the highest cerebral centres as a reflex mechanism is utterly contrary to the views of Jackson, who held that the highest centres, while more complex than the lowest, are, like the lowest, subject to the laws of reflex action. Thus he wrote (II, 63):

'A man, physically regarded, is a sensorimotor mechanism. I particularly wish to insist that the highest centres—physical basis of mind or consciousness—have this kind of constitution, that they represent innumerable different impressions and movements of all parts of the body, although very indirectly, as certainly as that the lumbar enlargement represents comparatively few of a limited region of the body nearly directly. . . . If the doctrine of evolution be true, all nervous centres must be of sensorimotor constitution. *A priori*, it seems reasonable to suppose that, if the highest centres have the same composition as the lower, being, like the lower, made up of cells and fibres, they have also the same constitution. It would be marvellous if, at a certain level, whether we call it one of evolution or not, there were a sudden change into centres of a different *kind* of constitution.'

The purpose of this paper is to demonstrate some concrete applications of Jackson's views on the reflex constitution of the highest centres. Specifically, I shall try to show that certain mental symptoms may be looked upon as illustrations of what Jackson called 'reduction to a more automatic condition.'

While Jackson insisted that the laws of reflex action apply to mental

* Bracketed figures refer to volume and page of the *Selected Writings of John Hughlings Jackson*.

phenomena, yet, disclaiming any knowledge of psychiatry, he attempted only in the most fragmentary way the task of applying these laws to the study of specific mental symptoms. Therefore, while I believe the formulations offered in this paper are in accord with his views, I cannot with certainty say he would have sanctioned them, and it would be a mistake to charge him with any errors and crudities to be found in them.

In accordance with Jackson's teaching (II, 63, 72, 156, 160), a distinction will be made, throughout this paper, between mental states and nervous states. The brain is a material thing; the mind is something immaterial. During every mental state there is a concomitant nervous state. The two should be considered separately in any attempt to analyze the nature of mental processes. The study of the immaterial thing, mind, is psychology; the study of the material thing, the brain, is physiology. As physicians, it is not enough that we consider mental symptoms as mental phenomena (a psychological study); we must also consider the nervous processes concomitant therewith (a physiological study).

The nervous system may be regarded as a complicated series of reflex arcs, superimposed one on the other. The lowest are the least, while the highest are the most, complex. Jackson roughly divided the central nervous system into lowest, middle and highest centres, but he emphasized that each of these levels may be divided and subdivided into lower and higher portions, so that really the central nervous system is made up of an almost infinite number of levels. Following Jackson's terminology, I shall speak of the highest 'centres,' which are, in turn, divided into lower and higher 'layers' (I, 380; II, 50, 56, 437, 480).

Lower levels of the nervous system differ from higher levels in two important respects: they are less complex and more automatic.

1. *Complexity.*—The gradation from lowest to highest centres is a gradation from least to greatest complexity. Consider, as an example, two problems in mathematics—problem A, which can be solved by the average man, and problem B, which can be solved only by the most eminent mathematicians. Obviously B is more complex than A. Moreover, the nervous substrate utilized in comprehending and solving B is more complex than that for A. When an eminent mathematician is in process of solving B, the very highest layers of his highest centres are engaged; when he is solving A, the layers engaged are in some degree lower.

2. *Automaticity.*—The gradation from lowest to highest centres is also a gradation from most to least automatic (II, 46, 53, 68). A reflex arc may be termed automatic to the extent that it functions stably under a variety of conditions, without requiring the attention of the subject. Thus the reflex arcs engaged during walking are more automatic than those engaged during the solving of a difficult mathematical problem: one can walk without attending to it, but one cannot solve a difficult problem without giving it one's closest attention.

The nervous system, then, may be regarded as an elaborate structure of reflex arcs, varying (as we proceed from below upward) from the least complex and most automatic to the most complex and least automatic.

In studying examples of mentation, one must consider not only a particular mental act but also the situation which evokes it. In the following example I shall consider two situations which evoke a response consisting of the utterance 'Good morning.' (*a*) On the one hand, one may meet a friend, and, returning the friend's greeting, may say 'Good morning.' (*b*) On the other hand, one may be conversing with a foreign acquaintance who is trying to learn English; the foreigner may ask, 'What do people say as they greet each other in the morning?' and the reply will be 'Good morning.' The utterance in (*a*) is the same as in (*b*), and one might be tempted to say that the nervous pathways engaged in both cases are the same. Actually this is only partially true. Both utterances are responses to stimuli, and since the stimuli in (*a*) and (*b*) are different, the reflex arcs engaged cannot be identical. The two reflex arcs coincide only in so far as they have a 'final common path.' The difference in the two arcs may be stated thus: the arc engaged in (*a*) is more automatic than in (*b*). Saying 'Good morning' in response to a greeting is a highly automatic act: one may say it even when one is deeply absorbed. On the other hand, saying it in reply to the foreigner's query is less automatic, since some degree of attention is required. Here, then, we have two reflex arcs, one slightly, the other highly, automatic, sharing a final common path. (This viewpoint is further discussed in the next section.)

Considering further the last example, it is conceivable that a lesion of the highest layers of the highest centres might extend downward just far enough to interrupt the reflex arc utilized in (*b*) without interrupting that utilized in (*a*). In such a case the patient would be unable to reply 'Good morning' to the foreigner's query, but would be able to say it in response to a greeting. There would, in other words, be loss of the more complex utterance of 'Good morning,' together with retention of its more automatic utterance. Such 'selective' loss of function may seem incredible, but really is not. In the next section I shall give actual instances of this type of loss.

The following proposition, then, is the keynote of this paper: paralysis of the highest layers of the highest centres may produce loss of the least automatic (most complex) mental functions, while leaving unimpaired those mental functions which are more automatic (less complex). This retention of more automatic function is what Jackson implied by his phrase 'reduction to a more automatic condition.'

How can one distinguish the more automatic and the less automatic activities of the organism? One criterion I have already alluded to: a function is automatic in the degree in which it may be exercised without the subject's attention. The only other criterion to which I shall here refer relates to priority of appearance in the ontogeny of the organism. Those

functions which appear first become the most automatic—a proposition in keeping with the fact that, speaking generally, the nervous system develops from the bottom up. Children can walk long before they can solve problems in geometry. This means that there is a stage at which the substrate of walking is fully evolved, while the substrate for the solving of problems in geometry is not. Anatomically, the latter substrate occupies a higher level than the former. A lesion of the highest centres might interrupt the latter and not the former, in which case there would be loss of the more complex function, mathematical reasoning, and retention of the more automatic function, walking.

It should be noted that I do not say that in *all* cases of nervous disease there is loss of the most complex functions and retention of the most automatic. I say this is true only of those cases where the dissolution is from the top downward. It is not true of cases where the lesion is confined to parts below the top. Thus, a patient with tabes and with intact highest centres will show impairment of some of his more automatic functions, such as walking, but no impairment of his complex functions, such as the ability to solve problems in geometry. This combination of phenomena would never occur in cases of disease of the highest centres.

Before proceeding, the terminology to be used in this paper will be further defined.

1. For each function and for each mental state there is a nervous substrate. The ' substrate of walking ' denotes that combination of reflex arcs which is engaged during walking. When one sees a brick, one has what Jackson called a ' vivid image ' (II, 70). At this moment there is activation of many reflex arcs, comprising what may be called the ' substrate of the vivid image " brick." ' When one merely thinks of a brick, one has what Jackson called a ' faint image ' ; the reflex arcs now engaged may be said to comprise the ' substrate of the faint image " brick." '

2. The adjectives ' automatic ' and ' complex ' will be applied both to functions and their substrates. Thus, comparing the functions walking and thinking, we may say that the former is the more automatic (or the less complex) ; we may say the same of their respective substrates.

3. Functions and their substrates are automatic only in a relative sense. Thus it means nothing to say that walking is an automatic function. Walking is more automatic than abstract thinking, since it appears earlier in life. On the other hand, walking is less automatic than grasping, since the former appears at the age of one year while the latter is present at birth. The relativity of the concept ' automatic ' may be compared with that of the concepts ' tall ' and ' short.' For the sake of convenience some men are called tall and others short, but really every man has some degree of tallness. A dwarf measuring 2 feet in height has 2 feet of tallness. When we say a man is tall, what we imply is that he is taller than most other men. Similarly, when one speaks of some functions as automatic, it should be remembered

that all functions are in some degree automatic. Even the functions of the highest cerebral centres are automatic, though relatively slightly. Sometimes in order to avoid cumbersome phraseology I shall say of a function merely that it is automatic, when I mean that it is more automatic than some other function under consideration.

4. In addition to 'automatic' and 'complex' the words 'voluntary' and 'organized' are sometimes used, 'voluntary' being applied to functions and 'organized' to their substrates. For example, thinking is a more voluntary function than walking; this is another way of saying that thinking is more complex than, and less automatic than, walking. Speaking of their substrates, we may say that the substrate of mentation is *less* organized than that of locomotion; again this is another way of saying that the substrate of mentation is more complex than, and less automatic than, that of locomotion.

In the first part of the last sentence the word 'less' is purposely emphasized. Physicians sometimes say that the highest centres are *more* organized than the lowest. The highest centres are said to be 'highly organized,' while the lowest are 'unorganized.' Thus one writer spoke of inhibiting influences which the highest centres exercise 'over automatic and unorganized motor processes.' Another, referring to Jackson's three levels, spoke of the highest level as the 'third and most highly organized level.' This is exactly opposite to the way Jackson used the word 'organized.' Jackson spoke of the lowest centres as the *most* organized, and the highest as the *least* (II, 395, 437). The most organized centre is that in which the connections between the component pathways are most stable, most resistant to interruption. It is that which functions stably under the widest variety of conditions. On this view, the most organized centres are the lowest. Thus the centres of circulation and respiration are so highly organized that they function stably day and night unceasingly until death. By contrast, the highest centres are so slightly organized that their smooth functioning is impaired by very slight influences: a little alcohol or a moderate degree of fatigue will prevent one from exercising his reasoning powers to their fullest extent.

After these preliminary remarks, I shall now consider the significance of Jackson's views for certain specific mental symptoms.

LOSS OF VOLUNTARY, AND RETENTION OF AUTOMATIC, PERFORMANCE OF CERTAIN ACTS

Jackson repeatedly pointed out that in aphasia it is not words which are lost but certain uses of words. Taking as an example the word 'No' (one of Jackson's favourite examples), there are three ways in which the word is used (II, 134). (1) A man may angrily shout 'No!' when his child is about to do something bad, or anxiously when the child is about to make a misstep, or incredulously on hearing some astonishing bit of news. These illustrate the 'emotional' use of the word. (2) He may say 'No' in reply to a question

requiring a negative answer. This is the ' propositional ' use of the word. (3) He may say ' No ' in response to the request to say it. Jackson described this use of the word, but did not name it. For the sake of convenience I shall refer to it as the ' volitional ' use of the word.

Jackson observed that in aphasia the volitional use of ' No ' is most impaired, the propositional use less so, and the emotional use least of all. Thus in one case (II, 134, footnote ; 177) the patient could not say ' No ' when asked to do so, yet said it in response to the question ' Are you ninety years old ? ' and shouted it angrily when his child misbehaved. Jackson's other examples need not be cited here. The point which is important here is that in aphasia, as Jackson emphasized (II, 49, 160), the uses of words are disturbed inversely as their degree of automaticity, the most automatic uses being least disturbed, the least most.

It would be wrong to suppose that there is only one substrate for the utterance of ' No.' If that were true, a lesion which interrupted this substrate should abolish the power to say ' No ' under all circumstances. In reality (if we consider not merely the actual utterance of the word but also the situation which evokes it) there are innumerable substrates for the utterance of ' No '—as many substrates as there are situations which evoke the response ' No.' These substrates may be grouped into those for the emotional, those for the propositional, and those for the volitional, use of the word. All of them have a final common path, consisting of those portions of the lowest centres which must be activated in order to permit the actual vocalization of the word. Before converging into their final common path, these substrates occupy diverse levels in the higher centres. The ' emotional substrates ' (substrates for the emotional use of ' No ') are the most automatic and occupy levels lower than the others ; the ' propositional substrates ' are less automatic and occupy higher levels ; the ' volitional substrates ' are the least automatic and occupy the highest levels of all. A lesion of the highest centres will impair the ' volitional substrates ' first and most, the ' emotional substrates ' last and least.

It may seem pedantic to insist on considering the substrate of the stimulus which evokes the utterance ' No ' as well as the substrate of the utterance itself. To show that it is neither arbitrary nor pedantic but, on the contrary, consonant with good neurological reasoning, I shall refer to the selective loss of the pupillary reflex found in the Argyll Robertson pupil. Normally the pupil reacts to light and on accommodation. It would be absurd to say that since in both instances the result is the same—pupillary contraction—the pathways engaged in both must be the same. In reality, there are two pathways, one activated when the eye is exposed to light, the other when the eyes are accommodated for near vision. The two pathways coincide only in so far as they possess a final common path, which probably originates in the oculomotor nucleus. No one thinks it strange that there are cases in which the light reflex is lost while the accommodation reflex is retained. In

such cases everyone accepts the inevitable conclusion that there is interruption of the substrate of the light reflex but not of the accommodation reflex. The point of interruption of the substrate of the light reflex must be somewhere above the origin of the final common path which the two substrates have in common. When, on the other hand, there is interruption of the final common path, the pupil is irresponsive not only to light but also on accommodation. The contraction of the pupil may be compared with the utterance 'No.' There are innumerable situations which cause one to say 'No.' There are, in other words, innumerable reflexes whose common response consists of the utterance 'No.' Correspondingly, there are innumerable nervous substrates (reflex arcs) possessing a final common path. When there is interruption of this final common path, inability to say 'No' is complete, in the sense that under no circumstances can the patient say it. When, on the other hand, there is interruption of some of the substrates above the point of origin of the final common path, there is selective loss of the ability to say 'No': the patient can say it under some circumstances but not under others, just as in the Argyll Robertson pupil there is pupillary constriction on accommodation but not on exposure to light.

In two cases of delirium I have observed a phenomenon identical in principle with this selective loss of the ability to say 'No.' In these two cases the patient, being disoriented, failed to answer correctly when asked to name my vocation, *in spite of the fact that during the conversation just before the question he had spontaneously addressed me as 'Doctor.'* In one case (delirium in a chronic drinker with pulmonary tuberculosis and moderate fever), the patient, as I walked into his room at the Harrisburg State Hospital, said, 'Good morning, Doctor.' After a bit of casual conversation I questioned him as to his surroundings and found him disoriented; he thought the place was a factory in a small town near Harrisburg. Next he was asked what my vocation is; looking blank, he replied, 'I don't know *what* you do.' In the other case (bromide delirium in a patient with cerebral arteriosclerosis) I had the following conversation with the patient at the Harrisburg State Hospital. (You look pretty well this morning.) 'I can't say I feel worse, Doctor. I'm holding my own pretty well.' (Tell me, what place is this?) 'Isn't this a fair?' (What county is this?) 'Aberdeen County.' [There is no Aberdeen County in Pennsylvania.] (What State is this?) 'I really can't tell you.' (What is my vocation?) 'You must know that yourself, because I ain't employed for you to get that part out.' (What do you think my vocation is?) 'I don't know *what* your vocation is.' It may seem inexplicable that a man who has just addressed me as 'Doctor' should be unable to name my vocation, but it is no more inexplicable than the fact that sometimes an aphasic patient who has just replied 'No' to a question cannot say it on command. The explanation is the same. Spontaneously addressing a man as 'Doctor' in the course of conversation is an automatic act, like the spontaneous use of 'No' in conversation. By contrast, naming a man's

vocation on command is, like saying 'No' on command, a much less automatic (much more voluntary) act.

Thus far in this section only verbal acts (utterances) have been considered. Similar remarks may be made about non-verbal acts, such as movements of the face, tongue and limbs. There have been cases in which the patient lost the ability to perform a particular act voluntarily but was still able to do so automatically. Jackson (II, 153) alluded to an aphasic patient who could not protrude the tongue when asked to do so, yet protruded it after drinking, when, as was her custom, she licked her lips. Case 3 of Wilson and Walshe [7] was that of a woman with leftsided motor symptoms resulting from disease of the highest centres. One of her symptoms was inability to perform on request certain movements which automatically she performed quite well [7] (p. 218). Wilson and Walshe called attention (p. 223) to the occurrence of a similar phenomenon in a case reported by Goldstein, and (p. 224) in one reported by Kroll.

Particular reference may be made to hysteria, in which there are many instances of selective loss of more voluntary function. Thus some patients with hysterical aphonia can phonate while coughing, but not on attempting to converse or on being asked to say 'Ah.' Gowers [3] wrote (p. 914) that patients with hysterical aphonia 'have been known to speak in a loud voice during sleep.' The phonation of coughing and that of utterances made during sleep are highly automatic types of phonation; the phonation of ordinary conversation is less automatic; that of the response to the request to say 'Ah' is still less automatic. A perfect parallel exists between these patients and some of Jackson's aphasic patients. The hysterical patient, unable to phonate when asked to say 'Ah,' is like the aphasic who could not say 'No' when asked to say it. Unable to phonate in conversation, he is like the aphasic who could not say 'No' in reply to questions calling for a negative answer. *Able* to phonate while coughing, he is like the aphasic who was able to cry 'No!' as an expression of his emotion.

Patients with hysterical astasia-abasia may be unable to walk in ordinary situations, yet may be able to run perfectly in escaping from danger. In a fiction magazine I have read of an Italian soldier in the World War who, after a trifling injury, became completely paralyzed. The physician, suspecting hysteria, drew his gun and, in the patient's hearing, said it was a pity that the government in the interests of economy had decreed that all chronic invalids must be shot. As the physician aimed the gun, the patient leaped off the stretcher and ran away screaming with terror. The story was reported as having really occurred. I cannot vouch for its authenticity, but physiologically it is credible. If a fire broke out in a neurological ward, no one would be surprised to hear that hysterical paralytics ran to safety. Cases of this sort, in which a helpless paralytic is able to run in an urgent situation, have led some physicians to conclude that in hysteria there is 'nothing really wrong with the brain.' This untenable conclusion is based on the erroneous

belief that there is only one substrate of locomotion. In reality (if we consider not merely the physical movements of locomotion but also the situations which evoke it), there are as many substrates as there are situations capable of evoking a locomotor response. These substrates converge in a final common path, but before converging they occupy varying levels in the central nervous system, the level depending on the degree of automaticity of the locomotor response in a particular situation. In situations of peril, locomotion is a highly automatic function ; in the ordinary situations of life, it is a more voluntary function ; during a physical examination, when a person walks in response to the physician's command, it is even more voluntary. A patient's ability to run from a fire when in ordinary situations he is helpless by no means signifies that there is nothing wrong with his brain. It does not even signify that there is nothing wrong with the substrate of locomotion. All it signifies is that there is nothing wrong with the substrates of automatic locomotion, the pathological process affecting only the substrates of voluntary locomotion. The pathological process, in other words, is high rather than low in the central nervous system.

THE EVOLUTION OF THE HIGHEST CENTRES ; THEIR ACHIEVEMENT OF INDEPENDENCE OF LOWER CENTRES ; LOSS OF THIS INDEPENDENCE THROUGH DISEASE

This section is a continuation of certain parts of the discussion contained in my recent paper [5] on the psychiatric importance of Jackson's theories. In order briefly to summarize the pertinent parts of that paper, I shall refer to certain aspects of the rôle of the highest centres in mentation.

In discussing the physical processes concomitant with imagery, Jackson used the terms ' vivid image ' (e.g. when one actually sees an object) and ' faint image ' (when one merely thinks of, or visualizes, that object).

When one actually sees an object, the physical process, according to Jackson (II, 69), is as follows :

' I suppose that I am seeing a brick. . . . What first happens is that there is a peripheral impression (upon the retina), impulses then pass through the lowest, through the middle, and up to the highest sensory centres. . . . So far we have only stated one half of the reflex action, have only reached the physical condition in the highest sensory centres correlative with the colour of the brick. It and all other objects have shape, and this as much requires to be accounted for as the colour. The shape of an object is the relation of its several positions one to another ; our knowledge of this relation is by movements, in this case ocular movements. . . . By currents passing from the highest sensory centres, so to speak, " across " to the highest motor centres, and from these downward, through middle and lowest motor centres to muscular periphery, there is development of movements of the eyeballs. . . . Here we have . . . reflex action.'

When one *thinks* of a brick, the reflex action is confined to the highest

centres (II, 70) : ' The highest sensory and motor centres are alone engaged ; there is still reflex action, but only the central links of the great sensorimotor chain are engaged ; the central part only of the whole process which occurred in perception is done over again, and, the excitations being slight, the image arising is faint, and, the lower centres not being engaged, it is feebly and indefinitely projected, *seems* more part of ourselves.'

Assuming Jackson's views to be correct, we may say that the ' substrate of the *faint* image " brick " ' (the substrate activated when one thinks of a brick) lies in the highest centres ; it forms a component (the central or topmost component) of the ' substrate of the *vivid* image " brick." '

Turning from imagery to movement, we find a similar distinction between a movement actually made and one merely thought of. Quoting Jackson (II, 95, footnote) :

' When I actually move my arm . . . there is a process from highest motor centres, through lower centres, then by nerves to some muscles, which are discharged in a particular way. This is a purely physical process. . . . When we think of the movement, or remember it (popularly " have an idea of it "), the physical process is limited to the highest centres ; *the very same* nervous arrangements of these cen'.es are engaged, but they are slightly engaged, and the psychical state concomitant with the slighter process is faint ' (italics in original).

According to Jackson, then, the substrate of an ' idea of a movement ' lies in the highest centres ; it forms the central or topmost component of the substrate of the actual movement.

In this paper I shall more particularly be concerned with a special instance of movement—speech. In sayin„ a word, e.g. ' tree,' one makes certain movements of respiratory and articulatory muscles. The nervous substrate activated when one says the word may be termed the ' substrate of the spoken word " tree." ' On the other hand, one may think of the word without saying it. The mental state now is a special instance of that which one has when one thinks of any movement ; it is an ' idea of a movement.' There is, concomitantly, activation of what may be called the ' substrate of the unspoken word " tree." ' The substrate of an unspoken word bears the same relation to that of the spoken word as the substrate of an ' idea of a movement ' bears to that of the actual movement.

Jackson's views on the nervous processes concomitant with faint images and with ' ideas of movements ' are of the utmost importance in a consideration of the nature of the substrate of mentation. Thinking involves, one might say, the ' interplay ' of faint images and ' ideas of movements.' Suppose today I am thinking of something I saw yesterday, e.g. an automobile accident. I visualize the scene, the paths of the automobiles, the collision, and I recall the sound of the crash and the cries of the injured. All these images that come to my mind are faint images. At the same time I have in my mind a series of unuttered verbal propositions describing the

occurrence : ' The two cars met at the corner, one travelling east, the other north. They were going fast and collided with a loud crash,' etc. Since I am only thinking of the accident and not talking about it, these words remain unspoken. During silent thinking, then, one may infer activation of substrates of faint images and substrates of unspoken words. Since these substrates lie entirely in the highest centres, it may be said that the nervous processes concomitant with thinking are (with trifling exceptions which need not be gone into here) confined to the highest centres. In other words, during silent thinking *the highest centres function independently of lower centres.*

But the capacity of the highest centres to act independently of lower centres is a trait which makes its appearance only after the highest centres have reached a certain degree of maturity. In early childhood the highest centres are relatively incapable of acting independently. When a young child thinks, the activity of his highest centres is accompanied by activity of lower centres. Since thinking is concerned with imagery and with movement, there are two manifestations of this infantile condition of the highest centres. (1) The child thinks in terms of *vivid* images, while the adult thinks in terms of *faint* images. (2) The child thinks in terms of *actual* movements, while the adult thinks in terms of ' *ideas* of movements.' These manifestations will be considered separately.

1. *Young Children Think in terms of Vivid, rather than Faint, Images.*— The evidence for this proposition was given in my recent paper [5] (pp. 862–864) and need not be repeated here in detail. To sum it up briefly, one may say that children are relatively incapable of representation ; they are, of course, capable of perception. Children therefore are relatively unable to appreciate the difference between ' I *saw* a dog running across the street ' and ' I *fancied* a dog running across the street.' (Ignorance of this characteristic of infantile mentation causes people unjustly to accuse children of lying.) The earliest representation is so weakly developed that it does not occur independently of perception. One sign of this is the habit children have of asking that a story be read to them in spite of the fact that they already know it word for word. If the reader should omit a single word of the story the child will correct him. The explanation for the child's wanting to hear the story when he already knows it is that he wants to enjoy the pleasure of thinking it through as a fantasy, but, being incapable of representation, he cannot *think* it save when he *hears* it. Thus the earliest representation is indistinguishable from perception, and only as the child matures do these functions become independent. This statement describes the psychological process. To describe the physiological process, one may say that in the infantile brain there are substrates of vivid images but none of faint images. The topmost portions of the substrates of vivid images form the rudiment of the future substrates of faint images. As the highest centres develop, these rudimentary substrates of faint images evolve, until at last they become fully developed substrates capable of activation even when lower centres are inactive. When

this stage is reached, the person is able to think even in the absence of auditory and other external stimuli.

2. *Young Children Think in terms of Actual Movements, rather than 'Ideas of Movements.'*—Mentation in everyone is intimately associated with movement, especially the movements of speech (words), but while adults think in terms of unspoken speech, children think in terms of spoken speech. A two-year-old child, looking out of the window, sees a lawn and trees, and people and a dog walking by; at the same time he delivers a continual chatter of words, phrases and sentences corresponding to the objects and events that he sees. This chatter is his way of thinking. The child learns many words and learns to form verbal propositions. To him, at first, these words and propositions are things to be *said*, not merely thought of. Only after much evolution does he acquire the ability to think of words and propositions without saying them. Speaking physiologically, in the infantile brain there are substrates of spoken words but none of unspoken words. (This is exactly comparable to the statement that in the infantile brain there are substrates of vivid images but none of faint images.) The topmost portions of the substrates of spoken words form the rudiment of the future substrates of unspoken words. As the highest centres develop, the substrates of unspoken words evolve, finally reaching the stage at which they are capable of activation even when lower centres are inactive. The child is now able to think of words and propositions without saying them.

The last paragraph dealt only with speech—a special instance of movement. The same remarks apply to all movement. Thinking of abstract matters is carried on largely in words (spoken and unspoken). Thinking of concrete matters, especially those involving one's own movements, is carried on partly in words (spoken or unspoken) and partly in body movements (actual or imagined). If near me I see an object which I want to hold in my hand, I have 'ideas' of certain movements—those movements of my upper limb which would enable me to seize the object. I 'reflect' that the imagined movements, if executed, would put me in possession of the object. But early in life I must have passed through a stage at which I could perform the movements but could not yet imagine them well enough to be able to indulge in this reflection.

To summarize: Of the manifestations of the evolution of the mind, we are particularly concerned with two. (1) The child is incapable, while the adult is capable, of representation. The images that enter into the child's thinking are vivid, rather than faint, images. To ignore the objects that lie within the scope of one's perceptions and to think instead of objects not present is an impossible or difficult task for the child. He can think of things only, or more easily, when they are presented to his senses. When he wants to think of a story—even if he knows it word for word—he can do so more easily if he hears it read to him (or reads it himself). From this he evolves into a person who can, when necessary, think of things not presented to his

senses. (2) The child is incapable of thinking without simultaneously *saying* (or otherwise doing) what he is thinking. From this he evolves into a person who, when necessary, can engage in deepest thought in silence and immobility.

This evolution of the infantile mind is made possible by a corresponding evolution in the highest cerebral centres, which, from being dependent on lower centres, incapable of activation save when lower centres are being activated, become at length so independent that now they may be activated when lower centres are idle.

Jackson, years ago, expressed the same conclusion regarding the independence of the highest centres. He wrote (I, 375):

'As evolution progresses the highest centres not only gradually develop (become increasingly complex, etc.), but also become more and more detached from, and more independent of, the lower centres out of which they have been evolved. . . . There are degrees of detachment and of independence. . . . (When independence has been attained) our highest sensory and highest motor centres (together the " organ of mind ") can energize, to a large degree, independently of the lower centres out of which they have been evolved, and by aid of which they have been developed; consequently they can act independently of the environment.'

Again, speaking of the hierarchy of nervous centres, he wrote (II, 477):

'The relations of these centres to one another is a very complex one. Besides relation of direction and control, the higher their evolution the more independent do centres become in their activity; there is quasi-detachment, so that in the most " detached " (the highest) centres activities can go on (internal evolution) largely independent of present converse with the environment; on the psychical side there can arise trains of thought independent of present experiences.'

The degree of independence attained by the highest centres depends in part on their healthiness. The healthier they are, the more independence do they attain. It is therefore to be expected that in acquired disease of the highest centres there would (at least in some cases) be some degree of diminution of the independence previously attained. Mentation in these cases would be characterized by either or both of two infantile tendencies: (1) the tendency to think in terms of vivid rather than faint images; (2) the tendency to think vocally rather than silently. Cases illustrating these tendencies will now be cited.

1. *The Abnormal Tendency to Think in terms of Vivid, rather than Faint, Images.*—This tendency, I submit, may be said to exist in all patients who have hallucinations. A hallucination is a vivid image occurring in a situation in which a healthy man would, at most, have only a faint image. Consider the case of a sailor who has on his forearm a tattooed figure of his sweetheart. When he looks at the figure, he may respond by thinking of his sweetheart, picturing to himself how she looked when he last saw her, and recalling what she said. These are faint images; they occur because the highest centres are

sufficiently healthy to be capable of independent activity. Now suppose the sailor happens to be in a delirium when he glances at the tattooed figure. The same train of thought will be evoked, but now it may be expressed in *vivid* images. The sailor may now have hallucinations of his sweetheart—he may ' see ' and ' hear ' her.

In the foregoing (hypothetical) instance we know the stimulus which led to the hallucination ; it was the sight of the tattooed figure. In most actual instances of hallucination the stimulus cannot be identified. Nevertheless these instances are open to the same explanation as the instance of the sailor. Believers in determinism will agree that each thought, or, more accurately, the cerebral process concomitant with each thought, is a reaction to the cerebral processes which existed a moment before. This principle holds even for those instances in which we cannot ascertain what thoughts (conscious or unconscious) immediately preceded the hallucination. I submit, then, that the following formulation, if valid at all, is of general validity. Supposing a series of cerebral processes leads to activation of the substrate of a particular idea, the form which this idea will take will depend on whether the highest centres are at the moment able to function independently of lower centres. If they are, there will be activation of highest centres only—activation of the substrates of certain *faint* images : the subject will *think* of certain things. If they are not, there will be activation of highest centres and lower centres as well—activation of the substrates of *vivid* images ; the subject will *see*, *hear*, or otherwise perceive these things.

In my earlier paper [5] I have discussed more fully the significance of hallucinations as manifestations of ' reduction to a more automatic condition.' I therefore pass on to the second manifestation of loss of independence of the highest centres.

2. *The Abnormal Tendency to Think Vocally rather than Silently.*—One of the popularly recognized symptoms of insanity is ' talking to oneself.' Everyone has observed some human derelict walking by on the street, muttering to himself. Here is a perfect instance of disease of the highest centres causing them to return to their infantile state. The demented patient, like the two-year-old child, *says* what he is thinking.

Some patients are painfully aware of this inability to think their thoughts without saying them. I [5] have mentioned the case of a woman who, in giving a retrospective account of a period of overtalkativeness following her emergence from a catatonic stupor, said, ' I had no speech control. It seemed as if I couldn't *think* things but could only *say* them. There was a great fear in my mind that I would never again be able to think without speaking, and I thought how horrible it would be to have to go through life that way.'

In the same paper I referred to Lindemann's [6] observations on the effects of amytal on normal subjects. Under the influence of the drug the following statements were made : ' It's funny I am telling you things which I wasn't

going to tell you. . . . No matter what comes to my mind it wants to be expressed too. . . . I feel like saying all sorts of things. The words kind of just come out of my mouth. . . . The words just go rambling on my tongue.'

The following additional cases have come under my observation.

A young woman, after recovering from a manic excitement, gave a retrospective account, in which she said that her chief trouble had been with her ' head running away.' In explanation of this phrase she said, ' You can't keep quiet. You can't keep your own secrets. Things that happened years ago would come to my mind and I'd say them aloud. I'd give everything away I had ; I couldn't keep quiet.'

It is possible that much, perhaps all, of the overtalkativeness of manic patients is a manifestation of inability to think without speaking. Manic patients *say* what healthy people merely *think*. I once walked through a ward accompanied by a very obese nurse. We met a manic woman who looked the nurse up and down and said contemptuously, ' My, how fat you are ! ' A healthy person in the same situation would have *thought* this proposition, not said it.

In the examples already given, the patient's thoughts were expressed in overt speech, so as to be audible to bystanders. In the next three examples the vocalization of the patient's thoughts did not reach this extreme degree.

A coal miner, age 31, entered the Harrisburg State Hospital suffering from schizophrenia. The two symptoms that will be mentioned here are hallucinations and a condition which he described with the adjective ' loud-minded.'

The hallucinations were auditory and olfactory. The auditory hallucinations consisted of voices which said vile things about the patient. In loudness they varied from a whisper to the volume which would be employed by a man calling another man ten yards away. They sounded like natural human voices, so much so that he steadfastly believed people were talking about him.

The feature of the case which is important here is the ' loud-minded ' condition. The patient first revealed this in giving an account of his illness, when he said, ' It all started when my brain got loud-minded.' (What does ' loud-minded ' mean ?) ' I mean, whatever I think, you can hear, without me saying it with my tongue.' (Can *you* hear it ?) ' Yes.' (Give an example.) ' Whatever I think, my voice-box goes like. I should think with my *head*, not with my voice-box.' (Give an example.) ' The other day I was wondering how my blood test was going to turn out, and I heard these words, " I wonder how my blood test is going to be." ' (Give another example.) ' I was thinking about getting parole here, and *that* got loud too : " Maybe they're going to parole me." My lips didn't say it, my voice-box said it. I think in my voice-box, and then it goes way up in my mind, and then it sounds just like—(gropes for the proper phrase)—transformed broadcasts.'

On another occasion he gave this description : ' If I was natural (i.e.

healthy), when I think of something I'd think just a little bit. *This* way, I must think with my voice-box. I must *talk* it—but not with my tongue. It's all in my head.' Because his thoughts were audible to himself and (he thought) to others, he spoke of them as 'loud thoughts.' They, however, were not loud in the ordinary sense: they were even quieter than a whisper. 'They don't sound like no voice: just like imagination. It's no voice. It just goes through me like—yet I can hear it.'

The patient drew a distinction between his 'loud thoughts' and his hallucinations ('voices'). The 'voices' impressed him as real: he thought people were actually making vile remarks about him. On the other hand, the 'loud thoughts,' he knew, arose in his own mind. Another difference is that the content of the 'voices' was usually expressed in the third person, while that of the 'loud thoughts' was always in the first person.

This, then, is a case in which the patient's thoughts, while unspoken, were accompanied by laryngeal sensations so intense as to lead him to believe his thoughts were being vocalized. He heard them himself, and thought others heard them too, though at the same time he realized they lacked the quality of ordinary utterances.

A woman of 61, with signs of cerebral arteriosclerosis, entered a moderately severe depression, one of her symptoms being that 'I talk to myself.' In describing this she said, 'Everything I think I'm going to do I say to myself. (For example) I "say" to myself, "Now I'm going to go to bed."' When out walking she 'talked' to herself about the things she observed. 'Almost everything I see I have to repeat.' For example, she 'said' to herself, 'I see this'; 'That's a car.' Many of the thoughts which she 'said' to herself were sensible: e.g. 'I don't know what to do'; 'I'm afraid.' On the other hand, some were strange and incomprehensible to her: e.g. 'I ask for punishment'; 'I refuse.'

This 'talking to herself' always occurred with lips perfectly motionless, so that members of the family were never able to observe anything unusual. She herself *heard* everything she 'said' to herself. It sounded like 'my own voice.' It was even quieter than a whisper. She knew that the phenomenon took place entirely in her mind.

A schizophrenic woman of 47 entered the Hospital, one of her complaints being that people 'seem to put thoughts in my head.' These thoughts she was compelled to 'repeat,' her lips being either shut or open and slightly in motion. Later, after much improvement, she said retrospectively, 'It seemed as though I had to repeat what they (hallucinatory voices) said. I repeated it in my mind, not with my lips—just like thinking it over.' The symptom had gone; as she said, 'I have more control.'

In the next two cases the patients did not think they were 'saying' or 'repeating' their thoughts, but mentation was accompanied by sensations referred to the organs of articulation.

A paranoid schizophrenic woman of 45 often heard a 'spirit' talking

to her. She identified the spirit as Jesus. She said, ' When I hear the spirit talking, I feel it in my mouth and throat. My lips and tongue move.'

A schizophrenic man of 22 heard voices which he termed ' undertones ' and which, he said, came in through his nose. When asked why they came in through the nose and not the ears, he replied, ' Because your mind is connected with your mouth '—a remarkable statement, considering the importance of speech (spoken and unspoken) in mentation.

Some of the foregoing cases show that there are degrees of loss of independence of the highest centres. To take but two instances : (1) the loss is great in those cases in which the patient's thoughts are actually vocalized, so as to be audible to bystanders ; (2) the loss is slighter in those cases in which the patient *thinks* he is vocalizing his thoughts when to bystanders they are inaudible.

Echolalia is a special manifestation of loss of independence of the highest centres. One of the results of our saying ' tree ' to a person is that he ' thinks of ' the word ; he has an ' idea of the word.' There is activation of the substrate of the unspoken word ' tree.' When the highest centres are insufficiently independent of lower centres, there will be activation of the substrate of the *spoken* word, with the consequence that on hearing the word ' tree ' the patient will actually *say* it.

The same explanation applies to echopraxia. When A claps his hands in the presence of B, there are two consequences in B. (1) B has vivid images (visual and auditory) corresponding to the sight and sound of A's movements. (2) He has an ' idea ' of A's movements ; he ' thinks of ' them. There are faint excitations in certain parts of his highest motor centres. When his highest centres are insufficiently independent of lower centres, B, instead of ' thinking ' of these movements, will actually execute them.

MASS REFLEXES IN THE HIGHEST CENTRES

In the stage of recovery of reflex function after complete division of the spinal cord, stimulation of any part of the lower limbs is followed by a widespread response consisting, in part, of flexor movements of the lower trunk and lower limbs. This has been called a ' mass reflex.' It typifies the response of the most primitive levels of the nervous system, released from the control of higher levels. It illustrates the general rule that the more primitive the nervous system, the greater is its tendency to produce widespread or diffuse, rather than narrow or discrete, responses.

Many further illustrations of this rule may be given. Coghill,[2] studying the earliest movements of *Amblystoma*, and Minkowski and others,[2] studying the movements of the human fœtus, have shown that in the earliest stages of the organism movements usually involve large segments of the body ; in later stages there is increasing occurrence of movements of small segments—movements of a single limb, or of part of a limb. When one compares an infant and an adult, the contrast is striking. The infant's movements are

unmistakably more diffuse than the adult's. As the infant matures, he becomes increasingly capable of movements involving smaller and smaller segments. The marvellously fine and precise finger movements of the violinist's left hand characterize the mature, rather than the immature, nervous system.

Examples will now be given to show the application of this general rule to the higher levels of the nervous system.

Becher [1] found that 'a large percentage of children, when told to open the mouth wide and put the tongue out, spread the fingers.' This is a forceful illustration of the occurrence of mass reflexes in the immature organism. In the nervous system of the young child, the substrate for opening the mouth wide and that for opening the fingers wide are inadequately differentiated, so that the command to open the mouth wide activates both substrates. Thus a relatively large cerebral area is activated by a stimulus which, in the adult, would activate a small area.

I have observed the following incident in a bright healthy boy 35 weeks old. The child, not yet able to sit unsupported, was being held by his mother while she sat in a restaurant eating a plate of soup. The mother held the child on her knee, and ate the soup with her other hand. Each spoonful, on its way to the mother's mouth, passed a few inches from the child's face. The child had had his own meal only two hours before, so that now he was not hungry. Nevertheless the odour of the soup aroused an alimentary response. After several spoonfuls had passed before his gaze, each succeeding spoonful was observed to elicit a response in the form of tasting movements. He watched the path of the spoon from the plate to the mother's mouth, and as it passed the point nearest his own mouth he made five or six tasting movements. The significance of this observation becomes evident when one considers what the response of an adult in a similar situation would be. An adult, when his appetite is aroused, responds only with salivation ; the infant responded with salivation (inferred, not observed) *and* tasting movements. In the infant's highest centres the area excited is larger than in the adult's. The infant's reflex, as compared with the adult's, is a mass reflex.

I now offer a similar observation in the case of a middle-aged man suffering from a fever delirium. The patient was in bed, wearing a hospital gown. When asked what time of day it was, he replied, 'I don't know—I haven't got my watch.' As he said this, he placed the fingers of his right hand into the right-hand pocket and then into the left-hand pocket of an imaginary waistcoat. A clear-headed man, if asked the same question while wearing a hospital gown, might have made the same verbal response, but no manual response. It may be objected that this example proves nothing, that the patient fumbled in his imaginary pockets merely because he was too confused to realize he was not wearing a waistcoat. This is a psychological explanation. Without going into the question of the merits and demerits of psychological explanations of behaviour, I say only that I am here trying to analyse the

physiological substrates of certain examples of behaviour. A physiological inquiry must deal only with nervous substrates and their excitation and inhibition. Viewing the delirious man's response physiologically, we must say that there was excitation of the substrates of a verbal reflex and a manual reflex, and that, in the case of a clear-headed man under the same circumstances, the substrate of the verbal reflex only would have been activated. The delirious man, therefore, showed, relatively speaking, a mass reflex.

I submit that the two symptoms discussed in the previous section—hallucinations and loss of the ability to think silently—are instances of mass reflexes. The substrate of a vivid image includes, and is therefore larger than, that of a faint image. When a psychotic man reacts with a hallucination (a vivid image) to a stimulus which in a healthy man would produce only an idea or a representation (a faint image), we may infer, in the psychotic man, excitation of a relatively wide cerebral area ; in the healthy man, excitation of a relatively narrow area. Similarly, in the case of patients who must speak their thoughts, there is excitation of a relatively wide cerebral area (the substrate of a spoken word being larger than that of an unspoken word).

Summarizing the data contained in this section, it may be said that the primitive nervous system is characterized by the relative predominance of mass reflexes—diffuse responses involving large parts of the nervous system. As the nervous system evolves, its parts become more and more complex, and more and more capable of acting alone, thus permitting responses involving smaller and smaller cerebral areas. When the nervous system is diseased, there may be a return to the more primitive condition, with the reappearance of mass reflexes.

AUTOMATIC REASONING

Automatic reasoning is illustrated by two examples. (1) Suppose one is asked to solve this problem in arithmetic : if *two* men can build *two* boats in *two* days, then *one* man can build *one* boat in how many days ? Stupid persons will say that the answer is obviously *one* day. This is an automatic response ; it is ' the first thing that comes to one's mind.' Only a person who has passed a certain point in cerebral evolution is capable of discerning its incorrectness. (2) This example consists of a trap which children set for each other. A asks, ' How do you pronounce M-A-C-C-A-R-T-H-Y ? ' B replies, ' MacCarthy.' A asks, ' How do you pronounce M-A-C-D-O-N-A-L-D ? ' B replies, ' MacDonald.' A then asks, ' How do you pronounce M-A-C-H-I-N-E ? ' B, if he falls into the trap, replies ' MacHine.' This too is an automatic response ; it is what the child will say ' without thinking.'

I do not mean to imply that all automatic reasoning is inferior reasoning. Reasoning may be highly automatic and yet in no way inferior. Suppose A asks B, ' I ate one apple, and I now have one apple left ; how many did I have at first ? ' B's response, two apples, is highly automatic, yet correct.

An automatic response is inferior only when it can be shown that another, more complex, response is better adapted to the circumstances. In this discussion I shall be concerned only with those automatic responses which are at the same time inferior.

Automatic reasoning is primitive reasoning. It is primitive because it characterizes those with poorly developed minds. It may therefore be assumed that automatic reasoning occurs during activation of lower nervous pathways, while more complex reasoning occurs during activation of higher pathways. Moreover, the lower pathways are activated before—even if only a split-second before—the higher. Thus a person solving the problem in arithmetic given at the head of this section might respond as follows: 'I suppose the answer is *one* day. . . . H'm, let's see, that doesn't seem right. *One* man can build two boats in *four* days; therefore he can build *one* boat in *two* days.' This response consists of two parts. First there is activation of lower pathways, producing an automatic, and (as it happened) incorrect, response; then there is activation of higher pathways, inhibiting the more primitive response and producing a more complex, and correct, response. When the highest centres are sufficiently underdeveloped or sufficiently paralyzed by disease, the automatic response is allowed to pass unchecked.

In the remainder of this section I shall discuss further the occurrence of automatic responses in 'normal' people, after which I shall give one instance of its occurrence in a delirious patient.

Responses which depend on suggestibility are automatic. It should be emphasized that positive responses to suggestion are not necessarily inferior. They are inferior only when they occur in circumstances in which they should have been inhibited. Thus when one gives a match to a friend who asks for it, one's obedience to the suggestion is eminently proper. But if a child of ten obeyed the suggestion to put his finger in the fire, his highest centres would fall under suspicion. Similar statements may be made regarding those responses which take the form of beliefs. Suppose on returning to town after an absence of several days I am told that yesterday it rained here. If I have no reason to doubt my informant, I respond by believing that it rained. This, an automatic response, is suited to the situation. On the other hand, when a known braggart says he is worth a million, it would be stupid to believe him automatically. Here the automatic response is checked by a more complex response.

Many of the beliefs of the vast majority of mankind are automatic beliefs—beliefs implanted by suggestion and maintained in the face of conspicuous lack of proof. For example, a woman spoke in glowing terms of a book of reminiscences by an astrologer who claimed he had foretold the date of many events, including the death of King Edward VII. She marvelled at this ability and said that until she read this book she had been unaware of the value of astrology. Her obviously sincere, and not ironic, manner showed that she had accepted uncritically the astrologer's statement of his achieve-

ments. In so doing she had reacted automatically in a situation which, in a more highly evolved person, would have evoked a more complex and more critical response.

An instance of an automatic response to suggestion in delirium will now be given. The patient, age 63, had a delirium associated with myocardial failure. In testing his orientation, I asked him to tell me the time. He pondered several seconds. Then, as it happened, the ward telephone gave two sharp rings. As soon as he heard them, he replied, 'That says two—I think it's later than that—I'd say it's about 3 o'clock—maybe 2.30.' (Actually it was 10.30 a.m.) The two rings had produced in him the mental state 'two,' whereupon uncritically he had accepted it as relevant to the topic of his cogitation. In a person with intact highest centres this mental state would have been promptly suppressed as irrelevant.

The lesson of these examples is plain. There are primitive and complex modes of reasoning. The former are acquired early in life, the latter late. The former occur during activation of lower layers of the highest centres, the latter during activation of higher layers. When the highest centres are incompletely evolved (as in children and in unintelligent and immature adults), or when disease (by interrupting the highest layers) reduces them to a lower level of evolution, there is 'reduction to a more automatic condition,' manifesting itself in relative predominance of primitive modes of reasoning.

MISCELLANEOUS EXAMPLES OF AUTOMATIC ACTION

1. The more one practises an act, the more automatic it becomes. To the greeting 'Good morning' one responds automatically 'Good morning'—automatically because one has done it so often. Sometimes circumstances arise which call for a response different from the habitual. In these circumstances the ability to break away from the habitual response is a measure of the evolution of the highest centres.

Children whom I examine address me sometimes with the polite forms reserved for women: they say 'Yes, ma'am' and 'No, ma'am.' Those who do this are usually below the average in intelligence, and usually come from poor homes. The reason for this behaviour is that children of poor families are accustomed to speak politely only to their school teachers (usually women). They rarely hear 'sir' and 'ma'am' at home and rarely are taught to use them, except at school. At school, when the teacher is a woman, 'ma'am' is used to the exclusion of 'sir.' In this way they acquire the 'ma'am' habit. Sometimes a child, after saying 'ma'am' to me, becomes embarrassed, having recognized his blunder, in spite of which—so hard is it to break the habit—he continues to address me in the same way.

The force of habit is seen in the case of those demented senile patients who give the year as 18 hundred and something. Taking an actual case, a patient of 75, seen in 1935, gave the year as 1835. This error indicates

the predominance of a more automatic over a less automatic response. Until the age of 40 the patient lived in the nineteenth century, and it was habitual with him to think of the year as 18 something. After the turn of the century it became habitual to think of the year as 19 something. The habit of mature years is less automatic, less stable, than that of childhood; it is the more easily lost—just as a language learned in later years is easier to forget than that learned in childhood. Accordingly, when the highest layers of the highest centres are diseased, there is loss of the more complex habit (19 something) and reappearance of the more automatic habit (18 something). There is a tendency to think that the errors of senile patients are haphazard. This is not entirely true. Errors in the last two digits of the year are sometimes seemingly haphazard, but errors in the second digit are always (in my experience) traceable to the strength of the habit laid down in childhood. When the patient errs in the century he always says it is 18 something. I have never heard a patient give the year as 17 something, because I have never had one who passed his childhood in the eighteenth century. For a similar reason I have never heard a patient give the year as 20 something. I think it probable that the senile patients of 100 years ago often made the mistake of thinking it was 1735, but never (or rarely) 1935. I also think it probable that the senile patients of 1890 never erred in the century; if this is true, it is because they never knew a time when it was not 18 hundred and something.

2. The untidiness of demented patients is automatic. In infants distention of bladder and rectum is followed promptly by evacuation. In older persons this response is suppressed save under special circumstances. Perhaps it will be said that the demented patient's untidiness needs no physiological explanation, that a psychological explanation is enough, viz. the patient is apathetic, and lacks the will to go to the toilet. But this is really no explanation; it is only another way of saying he is untidy. On the other hand, it *is*, I think, an explanation to say he is untidy because of impairment of those higher pathways which normally inhibit the primitive evacuation reflexes.

3. Some psychotic patients are easily irritated and respond belligerently with undue haste. An epileptic under my observation was sitting at his meal in the ward dining-room. To keep flies away from his glass of milk, he had laid a slice of bread on top of the glass. A fellow-patient came up from behind to borrow the sugar. In reaching for it he accidentally dislodged the bread from the epileptic's glass. Without pausing to ascertain the circumstances, the epileptic wheeled about savagely and for a moment seemed about to strike the other man. Such behaviour is automatic.

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Editorial

NEEDED REFORMS IN PSYCHIATRY AND NEUROLOGY

THAT there has been considerable progress in psychiatric work and education in this country in the last ten or fifteen years few will deny. But much more remains to be done. It is not that future progress need wait upon research —the knowledge and the precedents are there if we like to make use of them. But there are certain important points in which psychiatric practice is not yet abreast of the best continental and American standards.

There have even been some elements in the change of recent years which have not been altogether desirable. Alone of specialists among doctors, practitioners of the psychological aspect of medicine seem to find it necessary to lecture to sections of the public on the ultimate details of their specialty. It would not be thought desirable that a general physician or surgeon should expound the intimate working of the diseased organism; but it seems to be thought beneficial to dissect publicly the development of certain morbid attitudes of mind. This is a very doubtful policy, the more so as such lectures are apt to be attended largely by those whose curiosity is already morbid, or whose preliminary education along scientific and psychological lines is quite insufficient to enable them to listen to the lecture critically. It seems that lectures to the general public should be devoted not to itemized personal experience, but to general considerations such as the importance of healthy upbringing, of parental affection, reasonable education in certain matters at the right age, and knowledge of the earlier signs of mental illness. It would also be desirable to inform people where to go for reliable advice if nervous or mental illness appears to be developing or has already definitely

appeared, and to offer perhaps some elementary instruction in the attitude towards, and management of, those already ill.

This would be education of a useful kind and a source of information which the general public could assimilate without mental dyspepsia either regurgitant, bulimious, or flatulent.

The psychiatric education of doctors, both students and postgraduates, demands further attention. The recent curriculum conference focussed attention on the psychiatric education of the medical student ; but the recommendations fell short of what some at least think is indispensable. The addition of lectures on psychology in the preclinical period will, of course, help ; and the recommendation that clinical demonstrations should be less of mental hospital material and more of the commoner types and degrees of mental illness met with in practice is also wise. But nothing helps the student more in any subject than personal contact with patients. Undoubtedly the best thing to advance his psychiatric education is to see the individual patient at the out-patient department or the bedside. The latter will usually be out of the question until psychiatric clinics are built at general hospitals. The former could be arranged for a high percentage of, or in fact for all, students. At a large medical school with, say, a hundred students passing through per annum it would be possible to arrange that each student had approximately twelve three-hour sessions in the out-patient department. It is a pity that the curriculum conference did not recommend that this should be compulsory.

As regards postgraduate students, the consequences and implications of the diploma in psychological medicine have to be considered. At present the standard of general education and psychiatric information possessed by those passing the examination cannot be regarded as very high. The diploma has come to be looked on by many candidates, quite naturally, as a means of obtaining the extra £50 per annum awarded by most mental hospital authorities to those candidates who have the diploma. There is some evidence too that the diploma is open to abuse in that those who pass the examination feel naturally entitled to claim special knowledge of what might be called consulting quality, although the requirements

of the diploma as regards actual psychiatric experience are not great. It is worth considering whether the real value of the diploma should not be increased, so that anyone holding it must have had a standard of training in the basic sciences of psychiatry as well as in psychiatry itself considerably in advance of that professed by the majority of candidates of the present day. This requirement could be met by something equivalent to the tutorial system, requiring fairly close contact with teachers of psychiatry and others experienced enough to have a critical attitude towards the basic psychiatric sciences such as psychology. At present owing to the denial of facilities for study-leave a high percentage of the candidates are denied access to special tuition of this kind under such auspices for any length of time. But there will be great practical difficulty in raising the standard of postgraduate psychiatric education until the establishment at various university centres throughout the country of what has been advocated before in these pages, viz. psychiatric clinics including not only out-patient departments, but a considerable number of beds, as an integral part of the large teaching hospitals. Along with the establishment of such clinics should go the institution of chairs of psychiatry—and fulltime chairs preferably. It is an interesting fact that there is not a single such professorial post in this country. The Medical Research Council has lately been endowing psychiatric research more liberally; it is worth considering whether the funds available might not even more profitably be devoted to the endowment of one or two professorial chairs.

While in the diploma of psychological medicine the would-be psychiatrist must profess some knowledge of neurology, there is no corresponding converse arrangement for neurologists to make sure that they will profess some knowledge of psychiatry. Yet as a result of the popular misunderstanding of the term ' nerves ' the ' organic ' neurologist will continue to see a fair proportion of ' functional ' nervous conditions whether he likes it or not; and if he undertakes to treat these it seems desirable that there should be some method of ensuring that he is properly trained to do so. Again the solution seems to be the provision of psychiatric clinics in the medical schools so that proper facilities for adding in this way to his training

will be readily available to the budding neurologist. It would then become a matter of conscience that he should avail himself of these opportunities until such time as a diploma in neurological medicine may appear to be indispensable.

In taking a wide view of the reforms that are needed in psychiatry the conditions under which patients reach the psychiatrist, or reach mental hospitals, cannot be left out of account. The legal reforms in the Mental Treatment Act of 1930 have undoubtedly helped considerably, but they do not go far enough, as Professor G. K. Henderson pointed out in his recent Norman Kerr Memorial Lecture. We are still gravely handicapped by the conditions under which certain types of unstable person have to be dealt with. The legal requirements of certification at present are such that unless the patient is obviously mentally ill in cross section, as it were—that is, on interviewing him—it may be quite impossible to certify him. No real allowance is made under the existing law for the scientific aspect of the matter, and indeed scientific is synonymous in this case with the practical aspect, viz. the actual history of the patient. He may have shown himself incapable of living an ordinary life, and may be ruining his family—as, for example, the chronic alcoholic often does. It is obviously not only necessary for the welfare of his dependents but also for himself that some measure of control should be legally exercised over him. Even the method of signing a voluntary letter which is the necessary prelude to the admission of a voluntary patient to a mental hospital or licensed house is a sufficient deterrent to many. The mere existence of this proviso, which again would not be necessary if it were a matter of admission to a psychiatric clinic, accounts to a considerable extent for the large number of small mental homes conducted away from official auspices. Again, if psychiatric clinics existed as part of general hospitals there would be no necessity for having regulations for the admission of patients there in any way different from the regulations which govern admission to the other parts of the hospital. The present policy of the Board of Control is naturally to foster the interests of their own institutions and to advocate the building of admission wards in the grounds of mental hospitals. These are excellent in

themselves but cannot be regarded as in any way a satisfactory substitute for the psychiatric clinics here advocated.

There are other minor reforms which would tend to facilitate this treatment of mental illness, e.g. removal of the term 'certificate' from the 'urgency order' so that the socalled stigma of certification would be completely dissociated from the 'urgency order.' The latter could then be used as the Board of Control advocates it should be used, viz. as a means of securing observation temporarily until the matter can be definitely decided one way or the other.

Abstracts
Neurology

NEUROPATHOLOGY

[25] **The regeneration of the nervous system in the higher vertebrates** (La rigenerazione del tessuto nervoso nei vertebrati superiori).—O. Rossi and G. Gastaldi. *Riv. di pat. nerv. e ment.*, 1935, **46**, 1.

This is a very complete critical review of the subject extending to 323 pages and therefore incapable of proper abstraction.

It would appear that in the higher vertebrates nerve-cells damaged in their axiscylinders can reform these for a considerable length if the mutilation has not produced too great damage. Such a conclusion however should be modified by certain observations, otherwise the statement may be discovered to be erroneous in several respects. Regeneration is much less adequate in cortical, cerebellar and association neurones than in anterior and posterior root neurones. This may be due to the fact that the medium in which these cells lie is less favourable to regeneration, but it may also be due to some quality in the cells themselves.

When a protoplasmic process of a nerve-cell is injured this injury may be followed by an appearance of extensive and durable regeneration which however actually remains slight in extent. This may be due to the fact that the injury has affected the centres where regeneration is normally poor and of short duration. Thus in the case of the posterior root ganglia regeneration of the fibres conducting towards the ganglion-cell from the periphery is good, while regeneration of the fibres conducting away from the cell towards the spinal cord is poor. This difference must be due to some other cause than the simple capacity of fibres to regenerate. There is no doubt that an important part is played in the process of regeneration by non-nervous tissue closely applied to the nerve-fibres, viz. the cells of Schwann and certain glial cells. Such cells cannot however act unless the central continuity of the fibres with the cell is intact and the basis of regeneration resides in the cell.

Modern methods of study reveal that in the process of regeneration certain factors of a physical nature—such as rays of various sorts in the spectrum and of a more complex biological order, such as hormones in general, insulin, liver products, hæmatoporphyrin cell toxins, the action of which seems not sufficiently understood and not yet investigated—are important.

Clinical factors are however still of great interest since it is important to know the exact conditions favourable to regeneration of peripheral nerves and whether they vary in different regions.

Clinicians cannot remain indifferent to the still disputed questions as to the factors which favour or disturb the path of new fibres, factors involving the central nerve-stump, the scar, and the peripheral nerve-stump, since they have important repercussions on treatment.

Many clinicians have used a piece of nerve already advanced in degeneration and fixed in an appropriate fluid to bridge gaps in injured nerve. The idea of this is that the degenerated nerve allows an easy passage to the regenerating fibres. However, the fixation of the piece of nerve may produce changes which have a considerable effect on the process of regeneration since the vital characteristics of the piece of nerve are destroyed or changed. It is possible that a fresh piece of degenerated nerve which has not been fixed may be useful.

One of the most important branches of the subject is the possibility of grafting nerves in human beings so that fibres of a cut nerve can traverse the course of another nerve.

R. G. G.

[26] **A study of variations in the peripheral nerve-fibres at various ages and in different conditions** (Contributo alla conoscenza della fibra nervosa periferica in rapporto alle modificazioni che essa presenta in varie eta ed in varie condizioni).—A. Paleari. *Riv. di pat. nerv. e ment.*, 1936, **45**, 675.

The author has studied the changes in the peripheral nerve-fibres occurring as a result of senility, toxic states, circulatory disturbances, infective factors, etc., and also of histological manipulation in the course of investigation—which latter are sometimes regarded as pathological.

These causes, including the last, may be responsible for such changes in the axiscylinder as poorly staining areas, variations in calibre, even when quite conspicuous, neurofibrillar dissociation, simple interruption, and slight degenerative processes. The latter condition may well be produced by the laceration of the fibres in preparing the specimen. Similarly irregularities and interruptions in the myelin sheath may be artificially produced.

The author found that in persons over 40 there was often present in the cells of Schwann a substance which behaved differently with different reagents and therefore gave the impression of a pathological change.

In the connective-tissues within the nerve, round it, and in its sheath considerable variations both of structure and nature of the cells occur under the relatively normal conditions which have been investigated.

R. G. G.

[27] **Fundamental effects of epileptogenous agents upon the central nervous system.**—E. A. Spiegel and M. Spiegel-Adolf. *Amer. Jour. Psychiat.*, 1936, **92**, 1145.

The permeability of the cell-surfaces for electrolytes was studied in vitro and in vivo by measuring the polarizability of the brain tissue. Agents that

produce swelling of the brain by increasing the state of hydration of the brain tissue (e.g. distilled water, alkali) increase the permeability, as shown by the decrease in polarizability. This is a reversible process, as indicated by the effect of some acids and of hypertonic salt solutions after previous swelling of the brain. These experiments support the view that pathological processes which call forth swelling of the brain produce an impairment of the cell-surfaces in that their density is lowered. The transitory increase in permeability of the cell-surfaces, which is, according to modern theories of excitation, an essential part of the excitation process, is thus facilitated, and the threshold of the cells for metabolic or other stimuli is lowered. This explains the mechanism by which swelling of the brain increases convulsive reactivity.

C. S. R.

[28] **The Parkinsonian syndrome due to chronic epidemic encephalitis (von Economo type).**—M. A. Bahr. *Jour. Nerv. Ment. Dis.*, 1935, **82**, 514.

Two postencephalitic Parkinsonian cases without tremor have been studied clinically and anatomically. The most pronounced changes were found in the substantia nigra. Minor changes were present in the pallidum. There was a status cribratus in circumscribed areas of the striatum and in the cortex. In the white matter of the brain and in the globus pallidus and in the spinal cord a slight degree of diffuse demyelinization was present.

R. G. G.

[29] **Gliosis of the occipital lobes in the choreas.**—Charles Davison. *Amer. Jour. Psychiat.*, 1935, **92**, 361.

Fifteen cases of Huntington's and arteriosclerotic choreas were investigated. In eight of these, in addition to the usual neural structures involved in this disease, a dense gliosis was found in the white matter of the occipital lobe. Visual defects or hallucinations could not be demonstrated clinically. This type of gliosis was not observed in the white matter of the occipital lobes of normal individuals.

C. S. R.

SENSORIMOTOR NEUROLOGY

[30] **Instinctive, emotional and mental changes following prefrontal lobe extirpation.**—Spafford Ackerly. *Amer. Jour. Psychiat.*, 1935, **92**, 717.

A woman's left prefrontal lobe had been partially destroyed by a tumour, and her right prefrontal lobe had to be amputated in order to uncover and excise the tumour. Later findings of possible significance are here given. As regards the general intelligence level, there was marked slowness and

deliberateness with the Arthur performance tests. An apparent perseveration was noted, whether in the realm of ideas or activities, once the idea was formulated or the action initiated. There was an increased capacity for work and physical exercise without fatigue and no evidence of unusual mental fatigability, also a noticeable lack of anxiety about anything not of immediate concern to her, and, as well, more self-assertion. The patient flared up easily at her children and husband, but not at others. Also, according to her husband, there was a recent tendency to indulge in vulgarity—not previously seen. She carried out her usual household duties, but preferred 'quantity' production to 'quality,' though she was capable of the latter. The patient has learned to speak and read English better since the operation. The author concludes that in this case the brain, with possibly three-fourths of all prefrontal lobe destroyed, appeared to find difficulty in registering more than one stimulus at a time. This finding, he thinks, is not inconsistent with the conception that the frontal lobes enable us to become aware of an increasing number of simultaneous impressions.

C. S. R.

[31] **The problem of general as against focal symptoms in cerebral lesions: a contribution to general symptomatology.**—W. MAYER-GROSS and E. GUTTMANN. *Jour. of Ment. Sci.*, 1936, **82**, 222.

SEVERAL workers, dissatisfied with the theory of the localization of function in the brain, have put forward the view that all symptoms are due to one fundamental disturbance of a general nature. This, however, is not in accordance with the clinical facts. It is pointed out in this paper that even in diffuse brain lesions it is possible to discriminate between the different types of general disturbances. These types and the combinations between them are described.

A similar analysis is made of the socalled catastrophe-reaction of Goldstein, i.e. the response shown by patients with organic disease in situations which are too much for them. Various forms of this behaviour are described. Discussion of the relation between general disturbance of function and the catastrophe-reaction leads to a reexamination of the concept of focal symptoms. This concept is still found to be justified even in the case where general disturbances are caused by focal lesions; it is a problem of the interplay of focal symptoms. The psychiatric conception of syndromes is therefore suggested as applicable to the symptomatology of organic brain lesions.

C. S. R.

[32] **The function of the cerebellum from a clinical standpoint.**—K. GOLDSTEIN. *Jour. Nerv. Ment. Dis.*, 1936, **83**, 1.

AN important contribution in which the author seeks to simplify the conceptions of cerebellar function. In his view the special task of the cerebellum

is to support by coinnervation the performance of higher levels, namely, the voluntary purposeful performances which are determined by the whole organism, and automatically to maintain these movements and postures if the situation demands it, e.g. when attention is otherwise absorbed so that we are not able to innervate voluntarily. This coinnervation function is especially concerned with flexion and adduction performances.

In lesions of the cerebellum negative symptoms consisting in disturbances of supracerebellar performances particularly conditioned by the forebrain are found, and also positive symptoms—phenomena consisting of abnormal exaggerated reactions of the organism, particularly of the subcerebellar apparatus, to stimuli.

Various symptoms are discussed, but it is pointed out that when the cerebellum is out of action extension and abduction performances are exaggerated, and these are the reactions characteristic of responses to stimuli coming from the outside world. The subcerebellar centres are chiefly responsible for these reactions.

The failure to maintain uncomfortable postures in cerebellar lesions is due to the lack of support for the forebrain activity. Such lack of posture maintenance is specially noticeable when attention is diverted.

R. G. G.

[33] **Congenital arithmetic disability and acalculia (Henschen).**—Eric Guttmann. *Brit. Jour. Med. Psychol.*, 1936, **16**, 16.

The evidence pointing to the existence of congenital arithmetic disability similar to congenital reading disability is discussed. Recent observations indicate the occurrence of arithmetic disturbances (called by Henschen 'acalculia') after focal cerebral lesions. This symptom has been especially noted within the so-called parietal syndrome, occurring there with constructional apraxia, agraphia, disturbances of naming colours, etc. Such a case is described here. In some children with arithmetic disabilities, a similar combination of symptoms is demonstrable, and from this it is argued that cases may be regarded as specific arithmetic disabilities, arising from structural or functional anomalies of the brain.

C. S. R.

[34] **The occurrence of trigeminal neuralgia in patients having multiple sclerosis.**—J. M. Meredith and G. Horrax. *Jour. Nerv. Ment. Dis.*, 1935, **82**, 497.

Pain in multiple sclerosis and, in fact, neuralgia of any type are rare. Other sensory disturbances, such as numbness and paræsthesias, are comparatively common and probably occur at some period in the course of the disease in a majority of patients.

The pathogenicity of face pain in multiple sclerosis is unknown. The

theory that it may be due to plaques of sclerosis in the pons, medulla or the descending spinal root of the fifth cranial nerve is unlikely in view of the relief obtained by alcoholic injection or sensory root avulsion peripheral to these lesions. It is further refuted by the cases of multiple sclerosis reported in which lesions similar in type and location were discovered with no face pain whatever recorded in the history of the patient.

Twenty-five cases of concomitant trigeminal neuralgia and multiple sclerosis were encountered in a review of the subject in the literature of the last 17 years. In some the facial pain preceded the symptoms of multiple sclerosis, whereas, in others, the reverse was true. It is probably a fact that trigeminal neuralgia, complicated by multiple sclerosis, is more apt to be bilateral—not necessarily simultaneously so—than is the ordinary case of the former disease.

Two additional cases are reported in some detail, one of which had bilateral trigeminal neuralgia at different periods. Both of the cases were operated upon with subsequent complete relief of their pain for the length of time each has been followed (one year). There is no reason to believe that the pain will recur. Trichlorethylene inhalations and alcoholic injections were also effectual in temporarily relieving the pain in these two cases.

As far as can be determined, the modern methods of treating trigeminal neuralgia in the ordinary case are just as effective in the treatment of cases complicated by the occurrence of multiple sclerosis.

R. G. G.

[35] **A contribution to the clinical study of the pyramidal syndrome** (Contributo allo studio clinico della sindrome piramidale).—C. TRABATTONI. *Riv. di pat. nerv. e ment.*, 1935, **46**, 663.

THE author has made a clinical study of the pyramidal syndrome pursuing his researches in two directions, viz., (*a*) the value of pyramidal signs in the hand and (*b*) of crossed signs in hemiplegia. Fifty-seven patients suffering from hemiplegia or hemiparesis were examined. This was left-sided in 30 cases and right-sided in 27. Some normal individuals and patients suffering from other nervous diseases were also examined.

There is no reliable skin reflex in the hand corresponding to those in the feet.

The reflexes of Léri and of Mayer, and in a less degree the inverted Léri reflex, can effectively indicate by their absence or by the difference between the two sides the existence of a disturbance in the field of motor innervation of the upper limb. The signs of Klippel-Weil and of Wartenberg may occasionally be recognized on the paralysed side. Compared to those of the foot the reflexes in the hand are much less constant and much less in accordance with the severity of the lesion. In various neurological lesions the phenomena in the hand are not of any very significant diagnostic value.

With regard to crossed signs the author comes to the conclusion that disturbances of elementary movement can exist in the healthy side and are specially put in evidence by isolated movements of the affected limb. This is due to homolateral affection of the central motor neurone in right-sided hemiplegia in right-handed people.

R. G. G.

[36] **Cushing's disease and Recklinghausen's disease** (Morbo di Cushing e morbo di Recklinghausen).—C. Pero. *Riv. di pat. nerv. e ment.*, 1936, **47**, 183.

After reviewing the clinical and anatomical features of Cushing's disease, the author describes a case of dystrophia adiposogenitalis showing signs of Recklinghausen's disease as well, with osteoporosis, increased globulin in the spinal fluid, atrophic red marrow, but without hypertension or hypertrichosis. In this case hypercalcæmia with hypophosphatæmia and increased calcium in the urine were found.

The author points out that this picture of the mineral content of the blood is characteristic of the osteitis fibrosa of von Recklinghausen and that since this case as well as others described in the literature has clinical manifestations of Cushing's disease, he presumes there is a relationship between the two diseases and that they may be considered as one syndrome, specially characterized by the hyperparathyroidism of Cushing's disease. He discusses the doubts of himself and others as to the connexion between Cushing's disease and basophil adenoma of the pituitary. He finally discusses therapy.

R. G. G.

[37] **Medical and surgical aspects of Charcot joints.**—Samuel H. Epstein. *Amer. J. Syph.*, 1936, **20**, 386.

This article is a general discussion of Charcot joints, with illustrations from two neurosyphilis clinics. Emphasis is laid on the rôle of the orthopædic surgeon in the treatment of this condition.

Certain striking features of the disease are illustrated by the case-reports, which include an unusual Charcot involvement of the shoulder. In the author's experience Charcot joints occur for the most part in apparently stationary or arrested cases of tabes and the condition is not accompanied by any marked symptoms of tabetic disease. The local joint condition is usually the presenting symptom and these patients seldom come to the clinic for symptoms referable to disease of the central nervous system. Charcot joints are often multiple and patients develop a second or even a third arthropathy actually during treatment. It is an important fact that serological abnormalities in blood and spinal fluid are present much less frequently than in tabetics without Charcot joints. Antisyphilitic treatment alone is not

sufficient to check the progress of the joint disease. In every case cited orthopædic measures were finally resorted to, and in some instances successful functional results were obtained.

AUTHOR'S ABSTRACT.

PROGNOSIS AND TREATMENT

[38] **A new treatment of cerebral hæmorrhage and of its effects.**—R. COLELLA and G. PIZZILLO. *Jour. Nerv. Ment. Dis.*, 1935, **82**, 652.

AFTER having observed and studied numerous cases of encephalic traumatism, hæmorrhage, thrombosis, and embolism, in different periods of their progress, the authors have arrived at the following conclusions :

1. By some means not yet explained we may influence the focus of cerebral hæmorrhage and correct its effects to a considerable degree, and sometimes even totally, by intramuscular injections with the patient's own blood (autohæmotherapy).

2. The operation consists in withdrawing 25–30 c.cm. of blood from a vein of arm or foot, and in reinjecting it deeply into the gluteal region of the sound side. Before puncturing the vein, it is advisable to fill into the syringe a few cubic centimetres of a 25 per cent. solution of citrate of sodium, in order to prevent a premature coagulation of the blood.

3. The intramuscular autohæmotherapeutic injections are hæmostatic and useful for curing cerebral hæmorrhage and its effects in all cases, whatever the origin of cause of the hæmorrhage, whatever the patient's age, and the time of attack. The beneficial effect varies in degree, but is constant.

4. Recovery is to be observed even in most acute cases, especially in those of traumatism of the head with genuine cerebral hæmorrhage. The effects will be the better, the sooner curative intervention is attempted.

5. Autohæmotherapy helps to cure cerebral hæmorrhage before, during, and after ictus. It is most indicated as a preventive cure in cases of arterial hypertension with predisposing hereditary conditions ; for arteriosclerotic patients who often preannounce ictus by vertigo, debility of limbs, unilateral tremor of extremities. These effects are avoided and immediately corrected after the injection, which suddenly brings down intracranial blood pressure.

6. The blood injection allows a differential diagnosis between real cerebral hæmorrhage and cerebral softening. Although cerebral hæmorrhage is perhaps more frequent than softening and the symptoms are often identical, they may be distinguished because the curative effect is great with foci of cerebral hæmorrhage and nil with cases of cerebral softening.

7. Theories about hæmotherapy are numerous and vague. The nature of the action of these blood injections on circulation and capillary congestion in general and the reasons for the sometimes rapid disappearance of the symptoms are unknown.

R. G. G.

Psychopathology

PSYCHOSES

[39] **Relation of trauma to mental diseases.**—Abraham Myerson. *Amer. Jour. Psychiat.*, 1936, **92**, 1031.

Myerson has established for himself the following guiding principles: first, that wherever a psychosis is mainly of constitutional origin, and where medical science is yet unable to find any direct environmental relationship, he is reluctant to state that any environmental event is a probable exciting or precipitating or aggravating cause. If, however, an environmental event occurs which is of serious nature, such as severe trauma, or of great emotional significance, and if it can be shown that the psychosis occurred within a very short period, he is willing to admit some relationship. This is true, however, mainly of manic-depressive psychosis. He is willing to admit a psychological event as a probable cause for the occurrence of this psychosis. Manic-depressive is mainly an affective psychosis. It is therefore logical to assume that a serious emotional situation may, if immediately followed by a manic-depressive state, be a precipitating or aggravating cause. He is unwilling to admit that there is any reason to assume that this is a factor in the precipitation of dementia præcox, largely because the schizophrenic state is not, as far as we know, emotional in origin, and because the personality alteration is of more profound nature than the change which takes place in manic-depressive psychosis.

C. S. R.

PROGNOSIS AND TREATMENT

[40] **General paresis: treatment by tryparsamide-induced fever sequence.**—H. C. Solomon and S. H. Epstein. *Amer. J. Syph.*, 1936, **20**, 281.

This article gives a detailed analysis of 21 cases of general paresis, treated over a considerable period of time by tryparsamide, and subsequently given fever therapy. These patients were chosen for fever treatment from a series of cases treated by tryparsamide because they were not responding satisfactorily, either clinically or serologically, and they comprise the smaller number of therapeutically recalcitrant cases of a series that give a relatively high percentage of favourable results. The number of injections of tryparsamide given to this group varied from 28 to 301, and the average number of injections was 86. The time which had elapsed between the beginning of tryparsamide therapy and the fever treatment varied from one to ten years, with an average period of four years.

The results obtained after fever treatment were good, both clinically and serologically. The final analysis made one to several years after the fever indicated that none of the cases remained unimproved; three were considered as stationary; and 18 as arrested. In practically every case the cerebrospinal

fluid became negative, and in the majority of the cases the spinal fluid response occurred within 18 months following the fever treatment. These results make a marked contrast to those which usually occur in cases treated from the outset by fever. 'While a group of 21 cases may be too small a series from which to draw hard-and-fast conclusions concerning percentages, the practically universally excellent result emboldens us to conclude that a considerable number of preliminary tryparsamide injections greatly enhances the probabilities of therapeutic success in the treatment of general paresis by fever and that the improvement of the spinal fluid findings occurs more rapidly following fever if the patient has first been prepared by treatment with tryparsamide.'

AUTHORS' ABSTRACT.

[41] **Investigations on the problem of immunity against spirochæta pallida in general paralytics treated with malaria.**—A. BECK. *Jour. of Ment. Sci.*, 1936, **82**, 254.

IT is found that the serum and cerebrospinal fluid of general paralytics treated with malaria and of normal persons do not differ in their influence on the motility of spirochæta pallida; there is no evidence of agglutinins against the spirochætes of syphilis or of antibodies recognizable by the adhesion phenomenon (Brown and Davis). The virulicidic test on animals did not reveal the existence of spirochætocidins in the serum. No evidence was found of an antibody enhancing the phagocytosis of spirochæta pallida. The mechanism leading to the improvement of general paralysis is not of humoral, but of cellular nature.

C. S. R.

[42] **The hæmatoporphyrin treatment of melancholia and endogenous depression.**—J. HUEHNERFELD. *Amer. Jour. Psychiat.*, 1936, **92**, 1323.

A SUMMARY of the results of this therapy which takes into account only those who reported the number of treated cases and the degree of improvement shows that up to the present about 400 cases have been described. Of 327 cases here referred to 98 were distinctly improved, 162 cured or greatly improved, while only 67 remained uninfluenced. Of those who have made investigations only one or two obtained negative results, and in one instance one-half of the patients were schizophrenics, whereas there were strikingly favourable results with those who had involutional melancholia. All others confirmed the author's observations. Improvement first manifests itself on the somatic-vegetative side of the disease-picture and subsequently extends to the psychic field, in which the inhibitory symptom usually disappears first. It is thought permissible to ascribe to hæmatoporphyrin a specific influence on the melancholic and depressive conditions. Those whose disease-picture is

dominated by inhibition respond best. Among those cases which are influenced only slightly or not at all are to be found mostly those in which marked anxiety and lively hallucinatory elements predominate. In order to render the hæmatoporphyrin therapy still more efficacious, it will be necessary to attain a more exact demarcation of melancholic and depressive conditions.

C. S. R.

[43] **The value of insulin treatment in undernourished psychiatric patients.**—A. E. Bennet and Elvin V. Semrad. *Amer. Jour. Psychiat.*, 1936, **92**, 1425.

Twenty-five psychiatric patients on high caloric diets receiving insulin therapy for malnutrition were compared with 25 cases not receiving insulin, all other conditions being essentially equal. An average of 0·423 lb. per week excess gain was found in the insulin cases. The authors believe that this form of treatment is a definite adjuvant in the handling of the psychiatric patient with malnutrition. It should not be considered specific in any way, and the results obtained must be evaluated along with other operating therapeutic factors.

C. S. R.

[44] **Modern endocrinology and mental disorder.**—Thomas D. Power. *Jour. of Ment. Sci.*, 1935, **81**, 783.

It seems probable that all instinctive and emotional activities are accompanied by subtle alterations in hormonal balance, and that these in their turn influence neuronic processes. The close connexion between the diencephalon and the pituitary gland lends support to this view. It is easy to imagine how a profound emotional disturbance might be perpetuated by endocrine disharmony, and prove susceptible to substitution therapy. Stress is laid here on the part played by the pituitary in the development and activity of the reproductive organs, because it is felt that these physical processes have an important bearing on the force and vigour of the sexual instinct, which supplies the driving force of much of mental life. It is realised, however, that the dynamic side of sex involves more than a mere stimulation of the genital apparatus. Probably every hormone in the body plays its part in co-operating with this instinct, and what is wanted for dementia præcox is something which will produce a generalized stimulation of this nature. A combination of anterior pituitary hormones suitable for hypodermic injection would appear to be the ideal therapeutic agent, but until this preparation is on the market we must content ourselves with minor laboratory and clinical experiments.

C. S. R.

Reviews and Notices of Books

An Enquiry into Prognosis in the Neuroses. By T. A. Ross, M.D., F.R.C.P. Cambridge University Press. 1936. Pp. 194. Price 10s. 6d.

HARDLY anything has been written on this subject, so that a book which deals with the 'follow-up' for several years of nearly 1,200 neurotic patients who had been inmates of the Cassel Hospital for Functional Nervous Disease is certainly welcome. As the author says, it does not profess to settle the question, but it should stimulate further enquiries. The volume contains a mass of interesting and instructive information. The difficulties in obtaining after-histories that will give a correct indication of the condition are noted. Whether a permanent mental change can be effected in the neurotic and, if so, by what means and through what treatment, are vital points also discussed. We learn the very important fact that it is possible to effect a radical change in the mental outlook of many neurotics with permanent results by a short and simple therapy. Even long-standing cases can be thus successfully dealt with, and advanced age was not found so much a barrier as has been usually supposed. In many patients no exploration of the unconscious was needed and Dejerine's method of persuasion was frequently all that was necessary. Seemingly, repressions in the Freudian sense were often not found to be so causal as conscious ideas. The prognosis in special groups such as anorexia nervosa, traumatic and compensation neuroses, obsessional-compulsive neuroses, and alcoholism, is treated of in special chapters and brief consideration is given to the relationship of neuroses to suicide and insanity.

The second half of the book is devoted to statistical tables and case-histories. Every psychopathologist should study these pages carefully. The contents are not only authoritative, but lucidly and rationally written without that handicapping bias which is only too commonly found among members of some special school of thought.